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NUTRITION AND RELATED PROBLEMS.

ISAAC H. JONES, M.D., Los Angeles;
EUGENE R. LEWIS, M.D., Los Angeles;
and
HAROLD S. MUCKLESTON, M.D., Pomona, Calif.

Two decades ago came the great change in Otolaryngology. Practice and interest and the literature had largely featured surgery. Then came the sulfa drugs and then the antibiotics—and now our specialty is more in the medical than in the surgical field. Hilger,[1] however, considers that our opportunity has been broadened and that the prophets of doom have not spelled the end of otolaryngology. He feels that its scope is now enlarged by a closer integration of our field with general medicine — that the otolaryngologist has become more of a doctor and less a narrow and isolated specialist.

Interest in nutrition is a symbol of this trend. Likewise studies of the endocrines, autonomic influence, stress and allergy.

To some nutrition simply means food; if, however, it is defined as the sum of all processes by which the body is nourished, or as the sum total of metabolic processes, both anabolic and catabolic, which result in the health or disease of tissues, or simply as "what accounts for the health of tissue, good or bad" — nutrition might actually be considered to include these other related subjects. At any rate nutrition, endocrines, autonomic influence, stress and allergy are all

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interwoven inextricably in clinical practice. It seems to us that they not only overlap but often interlock — one dependent on the other. The understanding of one is certainly not clear without an understanding of the others. Each of these subjects is elaborately presented in the literature, *separately*, but one wonders if each might not be considered as a finger and all five might not be considered together, as the thumb and fingers of a hand. Into each one's practice comes a hint of this interrelationship and also the desire to master it. It is a consummation devoutly to be wished — although with such a vision comes the arresting thought that "if one does not aim beyond the stars he will not reach the stars".

Of all the problems in medicine is there no other that belongs with these "five fingers"? It would seem that the others, such as infection, neoplasm, trauma, heredity and congenital abnormality are not directly related to nutrition but are, so to speak, "on the other hand".

We find this idea helpful, not only with a patient whose symptoms have no obvious cause but particularly with those whose history and findings offer no clue whatever. We mention the five subjects and see if one or more of them may lead to something suggestive. At least it gives an approach to a problem; and in the discussion of these points the patient may reveal pertinent facts. For example, a woman has been greatly distressed, day after day for many weeks, by a feeling that she is about to pitch forward — never in any other direction. Vestibular findings normal in every detail. Cochleas normal. We then ask about nutrition and find that she has been studied at one of our best metabolic clinics for two weeks, with recommendation for increased protein. So that seems to be covered. Endocrines: Apparently negative. She had an uneventful menopause ten years ago without sequelae. Autonomic influence or allergy: Nothing in history or present state suggests either. Stress: For over a year she had worried greatly over her husband's incapacity to work — cause undetermined. This offered a possible lead toward

an explanation and relief and the idea was discussed with the patient. Following the husband's recovery the symptom promptly faded away.

Having offered some definitions of nutrition we will now consider some recently available data concerning the other four problems in an attempt to *correlate* them with nutrition as well as with each other.

ENDOCRINES.

As Mahoney[2] expresses it, the relation between the endocrines and otolaryngology could have been more clearly described several hundred years ago than today. The pituitary was then accepted as the source of nasal secretion and the thyroid as the source of the mucus of the larynx and trachea. In the words of de Schweinitz[3] "it was true then — because they believed it." Mahoney wisely regards endocrinology as a new field in physiology — even though he outlines the vast amount that is already known: (1) The anterior pituitary with its own five known hormones — all proteins. (2) The glands stimulated by the anterior pituitary — the thyroid, adrenal cortex, ovary and testis. (3) The posterior pituitary, pancreas, parathyroids, adrenal medulla and placenta. Like vitamins, their products are indispensable in nutrition. They derive from food their remarkable active principles which then in turn play such a large part in regulating the utilization of the food.

When vitamins first came upon the stage they held the center for years; their importance is in no way lessened now that they have found their place. At the moment it is the adrenal cortex that has gained great prominence and, like the vitamins, it is destined to maintain it. The unfolding story of the adrenal glands is presented by Mulholland.[4] For nearly 100 years it has been known that these organs, though so small, are essential to life. The modern story of the adrenal cortex began in 1930 when extracts became available for human use. The gland is concerned with the ability to exist in an environment which changes; its medulla provides epinephrin for sudden emergencies and its cortex provides the

many hormones for more profound environmental changes. These hormones from the cortex affect the electrolyte balance and the metabolism of protein, carbohydrate and fat. They have widespread effects in almost every metabolic process. Hyperactivity of the adrenal cortex causes marked diminution of eosinophils. The rate of their disappearance is fairly uniform; if 25 mg. of ACTH is given the fall is maximal in 4 hours. This is the basis of the Thorn test. With a competent and responding adrenal cortex the fall is from 50-100 per cent of circulating eosinophils. Sufficient evidence in the human being has accumulated to establish that the pituitary-adrenal cortex system is a protective buffer against environmental change.

AUTONOMIC FUNCTION.

Although he does not discriminate between functional variations and dysfunction Hilger[1] presents an ingenious interpretation. The very term "vasomotor", so often applied to the nose or internal ear, implies a motor mechanism; this is neural and centers in the hypothalamus which controls the autonomic functions of all parts of the body. It is influenced by the psychomotor areas in the frontal cortex and reflexly by body surface receptors. There is evidence that the pituitary is influenced — if not controlled — by the hypothalamus. It operates through the pituitary and hence through the blood; it also acts directly through neural pathways, to exercise direct cellular control in the peripheral tissues. The sole purpose of the autonomic nervous system is to contribute to man's adaptation to environment. Hilger's concept is that disordered function results when the hypothalamus is unequal to the task. The environmental influences are, in the order of their importance, emotional, physical, inhalant and ingestant. There first occurs arteriolar spasm; then ischemia, cell injuries and transudate. It becomes increasingly apparent that mucoid middle ears may result less from bacteria than from tissue reactions. In the bronchial tree neuromuscular activity may be added — asthma. Neurovascular change can affect the internal ear — cochlear or vestibular. Neural tissues tolerate ischemia poorly and lose function rapidly. Although

Hilger may seem to overemphasize autonomic dysfunction to the exclusion of other important factors, his concepts are pertinent and stimulating. In adapting his concept to the internal ear he[5] regards autonomic dysfunction as the prime factor in its many disturbances and endocrine imbalance as a frequent additional factor. He considers that the clinical picture varies with the neural segment involved and the degree of involvement; that chronic progression suggests minute neurovascular changes whereas extensive angiospasm accounts for complete loss of cochlear and vestibular function. He describes in detail the various medications which he used, the outcome in fourteen cases and his interpretations.

Many cochlear disturbances suggest to Cook[6] a mild hydrops which he terms a "water-logged" ear. The hearing fluctuates, at times the ear feels clogged up and sounds seem discordant or too loud. He finds this concept reasonable because in some the symptoms are transitory while in others they progress to deafness. A water-logged ear seems vulnerable to loud noises; he calls such persons "noise-susceptible." The majority of his patients were using salt in excess so he gave low sodium and high potassium intake for one month; if there was no response he added nicotinic acid as a vasodilator.

STRESS.

All who study the remarkable and elaborate work of Selye[7] may well be grateful to Guillemin[8] for presenting its main points. When any living organism is placed under abnormal conditions which disturb its vital equilibrium — in other words, under *stress* — the organism reacts against them. The phenomena which result are termed the "Alarm Reaction." It is a call to arms of the defenses of the body. These reactions which constitute the resulting shock are practically identical regardless of the nature of the insult; among other results, the experimental animal almost always develops gastric ulcer. In the countershock which ensues epinephrin is immediately thrown out by the adrenal medulla; then follows ACTH which causes the adrenal cortex to send out hormones of the cortisone type. This provides the organism with a

much more prolonged defense than that from the epinephrin. Also released are other endocrine substances not yet fully understood. Now, if the stress ceases this fight ends and endocrine hyperactivity subsides. On the contrary, if further stress is applied then the body must continue to defend itself against this abnormal condition to which it tries to *adapt*. Soon the organism reaches what is called the *stage of resistance*, in which it has developed an *optimal adaptation*. Now, if stress continues for several weeks, or even for several days if it is very strong, the defense mechanism collapses and the animal dies. These three stages — alarm reaction, resistance and exhaustion — are termed the *general adaptation syndrome*.

Ménière's Syndrome is generally regarded as due to hydrops of the internal ear. The observations of Fowler, Jr. and Zeckel[9] led them to the psyche as the main factor; the stress is occasionally from physical fatigue, illness or surgical operation, but is usually emotional in origin. They studied 23 patients with paroxysmal attacks of vertigo, nausea and vomiting, in addition to tinnitus and hearing loss. From the time of Ménière's original study in 1861 the lesion has been suspected to be vascular. The authors studied the vessels of the conjunctiva and noted clumping of red cells in the small vessels, before and after attacks of Ménière's Syndrome; extravasations and occasional small hemorrhages were observed. By analogy, such "sludged" blood in the internal ear could account for the symptoms and, if it lasted for any length of time, for endolymphatic hydrops.

ALLERGY.

It is refreshing to have Semenov[10] recall that not long ago — and yet the time seems distant — all rhinitis and sinuitis was attributed to infection; words such as "histamine" and "allergy" were known only to research workers; and Sir William Osler called syphilis "the great imitator". As matters now stand, the great imitator seems to be allergy. In any consideration of the pathology of the nose and sinuses we must reckon with allergy, not only its typical form such

as hay fever but also its masked form as in "collagen disease". Semenov notes that in some of these obscure conditions infection is the precipitating factor, but that the resulting storm rotates in allergy and often runs a relentless up and down course, which we fought for years under what he terms the "tattered and torn banner" of focal infection.

The hope that hearing loss may be prevented or restored by judicious antiallergic treatment is offered by Jones and co-workers.[11] For years many have regarded allergy as one of the causes of impaired hearing. As to the middle ear, the writers have observed permanent restoration of hearing by eliminating a common underlying cause of recurring nasal sinuitis, allergy. Recently interest has centered upon the theory that Ménière's Syndrome is vasomotor in nature, due to autonomic dysfunction or allergy. Helpful in treatment are elimination of contacts, a selection of foods which have minimal allergen content with maximal protein and complete nutritional values, and aid in the physical and mental rehabilitation of the patient.

Also in the experience of Shambaugh Jr.[12] allergy may be a factor in deafness, of both middle and internal ear types. The tube and middle ear are lined with the same respiratory mucosa that readily elaborates antibodies against foreign proteins and often develops clinical allergies. Allergy of the internal ear seems less easy to explain until we reflect that the endolymphatic tissues are derived from the ectoderm, which also readily becomes sensitive to foreign proteins. As in nasal allergy, allergy of the middle or internal ear is suspected when there are other allergic symptoms, when an aural discharge is very mucoid and resistant to treatment and when eosinophils are found in the secretion.

The line of thinking and recent emphasis on stress, autonomic influence and allergy has been applied by Phillips[13] even to multiple sclerosis. He notes that many consider allergy as playing a major rôle in etiology and he regards the personal, emotional problem of the patient as perhaps the

most important factor. He reasons that we are familiar with urticaria resulting from emotional and food allergy; having a common embryologic origin with the skin, it seems that tissue of the central nervous system may also respond with isolated areas of edema and pressure. Degenerative changes may or may not result. The cause of multiple sclerosis is unknown, but it does not occur in animals nor can it even be produced in animals. The most common symptoms are double vision and paralysis of an extremity but not infrequently one notes vertigo, headache, tinnitus and hearing loss.

Is there anything which favorably influences all types of allergic reaction? It seems to Rawlins[14] that from such a starting point it might be possible to develop a sound hypothesis of allergy. Cortisone may be an answer. His concept is that allergic edema occurs in the amorphous substance of subepithelial connective tissue. This mesenchyme contains hyaluronic acid which in normal individuals is a firm gel. The allergic reaction liberates many substances. Histamine has been regarded as the most important but Rawlins considers that hyaluronidase is also of importance, liquefying the firm gel and making it edematous. Cortisone affects this edema by neutralizing the hyaluronidase. This hormone, then, is an antihyaluronidase factor, as are also the antihistaminic drugs. In proposing this concept he does not overlook — in fact he emphasizes — the local autonomic dysfunction in allergy and the autonomic influence on the hormonal system.

* * * *

ITEMS ON NUTRITION.

Few of today's bits of news bear directly on ear, nose and throat practice but all have a direct bearing on ear, nose and throat problems.

This is our eleventh editorial on nutrition. Last year[15] we stressed the concepts of synthesis of vitamins within the body, and of antibiotics lessening this supply of vitamins as well as permitting yeasts and molds to flourish. We also

stressed that the creation of vitamins by bacteria in the intestine is obviously of great importance in nutrition — and that anyone who prescribes sulfa drugs or antibiotics is deeply concerned with their action on these bacteria. We said that the time was not yet when it could be stated that the sulfa drugs or antibiotics (1) lessen the supply of vitamins and (2) also permit yeasts and molds to flourish — but that there was a trend of opinion in that direction. The evidence indicated that (1) physicians, especially otolaryngologists, should be watchful and aware of these two new "Cautions"; (2) we should be more hesitant in giving antibiotics so freely, especially for trivial ailments; and (3) it would seem advisable to give B Complex with the antibiotic.

Since that time, new evidence seems to confirm these suggestions which we made with some hesitation last year. In a comprehensive survey Smith[16] calls attention to the complex balance which exists among microorganisms constituting the normal flora of the body. This balance is disturbed by antibiotics, with resulting vitamin deficiencies or actual new infectious disease syndromes. The importance of normal flora was not appreciated until it was disorganized by chemotherapy. In the struggle among themselves bacteria and fungi were employing their own antibiotics as offensive weapons long before they were discovered by man. Most of the known antibiotics which kill bacteria are produced by fungi; antibiotics derived from bacteria kill fungi, but unfortunately they are toxic for man. Following antibiotic therapy monilia are often found in serious infections of the mouth and pharynx, vagina and lungs; and fatal infections have occurred following invasion of internal organs. Although some intestinal bacteria may be destroying vitamins, most of them are engaged in synthesizing "K" and members of the B Complex group. The amount of absorption has been questioned but it seems probable that some vitamins are absorbed from the colon. Even a small supplement from this source may be of great importance.

Monilial infections of the mouth, intestinal tract, bronchi, lungs, skin and endocardium continue to be reported follow-

ing penicillin, aureomycin, chloramphenicol and terramycin therapy. The Lederle Laboratory suggested to McVay and Sprunt[17] that esters of paraben might prevent development of monilia which sometimes follows antibiotic therapy. The monilia flourish most frequently in the large bowel without serious consequence; however, deaths have occurred from moniliasis in other organs. These esters have been employed in foods and drugs as preservatives for many years. The methyl ester of paraben is effective against molds and the propyl ester against yeasts. The investigation used two preparations, one of aureomycin alone, the other of aureomycin with the paraben esters. Oral, vaginal and rectal studies were made on 186 patients. The results indicated that esters of paraben are of value in preventing overgrowth of monilia following aureomycin therapy.

We are reminded by Patton[18] that the chief causes of death and disability have always been violence, infection and degenerative disease. It is probable that infection dates from those forgotten years when man began to lead a communal existence. Against bacteria man has had little or no defense until a very short time ago; only within the last few centuries has any effort been made against them — beginning with quinine and the heavy metals. The deliberate creation of our modern drugs is a far cry from the development of quinine. In the 17th century Peruvian natives relieved a patient, sick of "intermittent fever", by infusions of a bark; and in honor of her husband, Count Chinchon, Viceroy of Peru, this bark came to be called "Cinchona". So its alkaloid, quinine, became the first "wonder drug". Later came vaccines and anti-sera; and the past twenty years have finally seen the direct attack upon bacteria by the sulfonamides and antibiotics.

It is not too much to say at the present time that, for acute conditions, perhaps half of all medical and surgical patients receive antibiotics as part of their treatment. Pulaski[19] lists seven as now commercially available. In surgery they are useful before and after operation. They are most effective in acute conditions characterized by cellulitis. Failure is not necessarily the fault of the antibiotic but may be explained

by (1) the use of the wrong antibiotic for a susceptible organism, (2) drug resistance (in streptomycin therapy), (3) inadequate dosage or (4) blind reliance on antibiotics as a substitute for surgery. Fleming said: "Antibiotics simply attack the bacteria — the surgeon must treat the patient."

It seems to Scholtz and Williamson[20] that dermatologists are on the verge of a great simplification in their specialty in that many disorders due to lack of "A" can be brought together as variants of the same basic disease — abnormal keratinization. They hail an advance in "A" therapy in that it can now be given in water instead of oil; results are more complete and more rapid on much smaller doses. Incidentally it is more palatable — having no fishy taste. Signs of the lack of "A" come slowly — and of reversal, equally slowly. Exophthalmos may result from excess of the thyrotropic hormone of the pituitary; thyroid extract helps in the rat, but not in man. The authors think highly of Barber's[21] recent concept of acne. The essential lesion is the comedone; it appears *pari passu* with pubic and axillary hair; these are known to depend upon stimulation of the pilo-sebaceous system by androgens; acne is "cured" by adequate doses of estrogen. The authors add what they regard as other factors in acne — hypothyroidism, disturbed "A" metabolism, anemia, physical fatigue and emotional stress. They recommend a high protein diet. The endocrines concerned with hair-growth are adrenal cortex, gonad, thyroid and anterior pituitary. True sexual hair in males is dependent upon the gonad; coarse hair in the external ear is seen almost exclusively in the male.

An extensive and helpful study of nutrition is given by Rosenvold.[22] Covering many contributions he applies them to everyday practice. From his own experience he confirms that the trouble is rarely due to lack of a single food factor — although he refers for example to improvement by "B_2" alone of seborrhea, fissures at the corners of the mouth and cheilosis. As to allergy, it is his belief that the nutritional approach will eventually overshadow the immunologic approach. He is impressed with the interrelationship between vitamins and

hormones. The use of endocrines must extend beyond the dispensing of thyroid extract; the utilization of proteins and minerals may require the prescribing of hormones appropriate to the individual patient.

Pollack[23] notes that certain oral lesions often develop in patients who lack protein; they resemble those of "B_2" or niacin deficiency — but massive doses of these vitamins may prove ineffective. Now, if enough protein is given, even with smaller doses of vitamins these lesions may rapidly disappear. In brief, lack of a vitamin may produce a lesion although there is good caloric and protein intake; but these same lesions may occur on an adequate vitamin intake in the absence of adequate protein. These interrelationships must be borne in mind. It is wise not to rely on one single sign as an indication of the lack of a specific nutrient. If the patient shows angular stomatitis and also cheilosis or facial seborrhoea or corneal vascularization or a magenta-colored tongue — a lack of "B_2" may be safely suspected. To diagnose "A" deficiency one must find certain changes in the eye and in the skin. "A" deficiency is noted chiefly in epithelial structures; ninety per cent of those showing this dermatosis also have eye manifestations. Lack of "B_2" may exhibit oiliness, greasy, flaky yellow scales and an erythema. There is some evidence of a relationship of acne to the B Complex. Certainly, several factors may affect a sebaceous gland, particularly "A" and "B_2". Lack of "A" may cause xerophthalmia, keratomalacia and nyctalopia. Giving large amounts of "A" may confirm these diagnoses. Lack of "B_2" may show at the angles of the mouth linear cracks radiating laterally; these open sores — perleche — if infected, may leave permanent scars. However, it is well known that often these lesions respond to "B_2" only if adequate protein is also given; the main lack may be in protein and the vitamin may serve only as a supplement. In examining the tongue daylight is preferable. The tip should project beyond the lip; a spatula is drawn over the dorsum from back to front. This is against the grain of the filiform papillae. Ruffling the papillae reveals the true status of their growth and the color of the underlying tissue.

True atrophy of these papillae not infrequently gives the appearance of hypertrophy of the fungiform papillae; conversely, hypertrophic fungiform papillae may be masked by the filiform if they are not brushed aside. A hand lens or binocular loupe may reveal changes not apparent to the naked eye. Normal gums are light pink, closely adherent to the teeth. The earliest signs of gingivitis are seen below the dental margins, a hyperaemic change and purple color; such inflammatory changes are easily differentiated from scurvy which, once seen, with its spongy, friable, bleeding appearance cannot be confused with anything else.

"C"

Even Hippocrates described scurvy and as Krolicki[24] notes, Kramer[25] gave citrus juices in 1720 and stated "you will, without other assistance, cure this dreadful evil". Many do not realize that "C" is found in all living plant cells and is also present in all animal tissues. Only guinea pigs, the primates and man are unable to meet body needs by synthesis of "C" and must get it from foods.

So much we know isn't so. What is a normal diet? As McLester[26] says, we must not be governed by too many rules. Man can nourish himself on widely different diets; in this he is very versatile. Robust health is found in the Himalayan mountaineers who live on fruits and vegetables; in Arab tribesmen who subsist largely on milk; and in Eskimos who eat only fish and meat. They all do well on what they are used to. The normal diet of a person of Italian extraction is entirely different from that of the Russian immigrant. It has been generally considered that one gets scurvy if he doesn't eat vegetables and fruit — "C" being the main requirement. Stefanson[27] spent ten years living as an Eskimo in the Arctic; when meat was not available they ate only fish month after month. He began to like it, even fish that was "high". At first he longed for salt which, prior to Columbus, had not been used in America; when he finally secured some he enjoyed it hugely for a time, but it soon palled on him and

when camp was moved he left the salt behind. Living on this salt-free meat and fish he, his crew and the Eskimos maintained perfect health; and there was no scurvy.

IRON.

The amazing response of pernicious anemia to liver therapy naturally led to its unbridled use in all types of anemia. Such shotgun therapy, which has been and still is so prevalent, is decried by Heck[28] who emphasizes that the treatment of anemia should depend directly upon the blood picture. He makes it clear when we should use iron and when liver; there should usually be no question as to which is needed. Most anemias fall into the iron-deficiency group, generally attributable to loss of blood, as in a bleeding lesion or menorrhagia. For such anemias liver extract, "B_{12}" and folic acid are definitely not indicated. Iron is usually given by mouth; if poorly tolerated it can be given intravenously. For pernicious anemia, in sharp contrast, the best treatment is liver extract or "B_{12}" parenterally. Folic acid is not needed. Iron is not indicated. In other diseases with megaloblastic anemia some patients do not respond to "B_{12}" but do improve on folic acid. None of these medicaments is helpful unless there is a deficiency of that particular substance. To this day vast sums are spent on iron, on liver extracts or on both together, with no regard whatever to the indications. All we need do is to determine the type of anemia; give only the indicated medicament; and not mix treatments.

CHLOROPHYLL.

This remarkable substance was a "pioneer" in all Nutrition. It provided plant life to feed earth's primitive animals — and all those since. It continues to be a *sine-qua-non* today: no chlorophyll, no plant; no plant, no animal.

In the atom bomb tests at Bikini and Eniwetok seeds of corn were exposed to radiation. These seeds were then planted and results observed.[29] There developed freaks of many types, including striking changes in the chlorophyll. Many of these mutants showed, instead of the normal green, a mottling from

pale or yellow to albino. Without normal chlorophyll the plant cannot utilize sunlight. Theoretically, 20 per cent of the light energy from the sun can be transformed into chemical energy by a plant and laid down as carbohydrate material. In reality, only 1 or 2 per cent of light energy falling on, say, a cornfield can be harvested as plant material.

Morgan[30] states that chlorophyll consists of two components of slightly different composition. It seems odd to Taber[31] that the first mention of its deodorizing effect was made only three years ago, by Wescott.[32] Chlorophyll was isolated in 1911; and introduced into medicine in the 1930s by Burgi,[33] who considered it helpful in various anemias. A product devised by him has been available in England for many years, yet its deodorant properties were not even mentioned. Wescott first noted relief from odors in the mouth — from food, beverages and tobacco; halitosis of unknown origin; odors from perspiration, foot, menses and urine. Serling[34] gave it to dogs and found that it eliminated objectionable odors within 24 hours. No one noted any toxicity or any effect on digestive activity. Consequently Taber studied the effect on menstrual odors, which are presumed to arise from decomposition of blood and cellular debris. It was doubted that chlorophyll could eliminate such odors, as they would appear to be beyond any metabolic influence — but the odors disappeared. The only speculation concerns the *modus operandi*. It is remarkable, but as yet no one has offered any explanation whatever. The skeptic shrewdly asks, "Why reeks the goat upon the hill — when all he eats is chlorophyll?"

Many clinical studies and reports have been made: Straubinger[35] has found that chlorophyll keeps wounds clean and promotes healing. He mentions that the iron radical in hemoglobin corresponds to the magnesium radical in chlorophyll; hemoglobin delivers oxygen and chlorophyll delivers CO_2. Horwitz,[36] likewise, states that both are concerned with oxygen-carbon dioxide interchange. There is this similarity in the physiologic action; and he suggests that this may have some bearing on chlorophyll's good effect in tissue repair. He thinks it should occupy a prominent place in dealing with

rectal infections and wound repair in general. Its value in the healing of wounds and burns has been reported by many. It has little effect on bacteria and its beneficial effect on healing may be exerted through stimulation of the cell. Incidentally foul odors disappear. Weingarten and Payson[37] gave chlorophyll by mouth and noted striking reduction of offensive odor in colostomy patients. Carpenter[38] found the local use of chlorophyll of definite value in treating 127 cases of chronic osteomyelitis and chronic ulcers. Moss, Morrow, Long and Ravdin[39] studied local effects on wound healing, odor and appearance. They noted that chlorophyll tended to produce a clean, granulating wound. Compared with cases not receiving chlorophyll this series showed a decrease in the odor of foul smelling wounds. Tremble[40] finds it useful in atrophic rhinitis with ozena. After removal of crusts a solution of chlorophyll is instilled three times daily. A 100 mg. tablet of chlorophyll by mouth after breakfast and dinner is usually sufficient to relieve odor. Also in cases of chronic otitis externa and media with foul discharge it cuts down the disagreeable smell.

There always have been areas of semi-starvation and others of actual starvation. Those who ever stop to think of it realize that the world's food supply is becoming less and less for more and more people. As Osborn[41] says, parts of the earth, once living and productive, are now dying at the hand of man. The time may come when our "plundered planet" will cease to supply enough food by the chlorophyll-plant-animal sequence. Man would then be compelled to resort to an ultimate though not impossible method of creating food directly from the air and sunlight, following the method of chlorophyll.

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INFLUENCE OF FENESTRATION SURGERY ON BONE CONDUCTION MEASUREMENTS.*

FREEMAN MCCONNELL, Ph.D.,
Nashville, Tenn.,
and
RAYMOND CARHART, Ph.D.,
Evanston, Ill.

Several investigators have noted improvement in hearing by bone conduction following fenestration surgery.[3,9] These observations are provocative, since the fenestration operation does not aim toward restoration of sensory-neural functioning. One possible explanation for the observations is that the structural changes produced in the middle ear by surgery had a mechanical effect which modified the patient's responsiveness to bone conduction stimuli. According to this view, the changes in bone response cannot be considered evidence that surgery improved cochlear reserve. Neither can preoperative bone thresholds be accepted as pure measures of "inner ear" sensitivity. The alternative interpretation is that bone measurements are sufficiently inaccurate so that the observed improvement following surgery was the result of chance and did not represent actual change in responsiveness to bone conduction stimuli. Such an explanation conforms to the opinion of many workers, who feel that bone conduction thresholds are direct, if somewhat inaccurate, measures of cochlear function.

The foregoing alternatives emphasize the need for further study of the effect which fenestration surgery has upon hearing by bone conduction. Such investigation can help clarify three matters of importance. First, it can contribute to our

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understanding of the interpretation to be applied to bone conduction audiometry by demonstrating whether or not mechanically induced changes in bone threshold do occur as a result of surgery. Second, if such changes do occur and their approximate magnitudes can be described, the surgeon will have at his disposal information which will help him to estimate with greater surety the amounts of inner ear involvement possessed by his candidates for the operation. Third, if a basis can be evolved for correlating surgical effects on bone response with postoperative improvement in air conduction acuity, the operating surgeon will have a further means of evaluating the adequacy of each operation through comparison of observed results with anticipated restoration.

These prospects gave rise to the experiment here reported. Its purpose was to establish more definitely whether or not changed bone conduction sensitivity results from fenestration surgery and, if it does, to determine the approximate amount and direction of change throughout the audiometric range.

— II —

The basic procedures of the study were simple. Detailed preoperative and postoperative audiometric tests were administered to 58 consecutive cases selected for fenestration surgery.* All tests were administered by one of the authors in the offices of the cooperating physicians. A clinical audiometer, the Maico E-2, was used to determine pure tone thresholds for both air and bone. Speech audiometry was performed by air with an Otarion Auditory Analyzer. Each patient was given two series of tests.†

A preoperative series was administered approximately two weeks before surgery, while a postoperative series was given about three and one-half months after the operation. The data thus gathered were later subjected to appropriate statistical analysis.

*These persons were patients of Dr. George E. Shambaugh, Jr., and his associates. They are, therefore, judged to represent a reasonable sample of the distribution of otosclerotics which competent otologists select for surgery.

†These tests were independent of those administered by surgeons for the purpose of determining the candidates' suitability for the operation.

— III —

Before considering the results obtained, it is pertinent to discuss briefly the controls which were instituted to insure adequacy of the audiometry. These controls were of two types: *1.* empirical confirmation that the test instruments were in satisfactory calibration, *2.* physical measurement of the performance of the instruments throughout the time span of the investigation.

Ten subjects with normal hearing were tested on the Maico E-2 Audiometer at the beginning and, again, at the end of the investigation. The average thresholds thus obtained for the various test frequencies are shown in Figs. 1 and 2. The solid line in each figure connects the averages obtained at the beginning of the investigation, while the broken line connects averages obtained at the conclusion. Fig. 1 summarizes the data for air conduction, and Fig. 2 shows the bone conduction results. One notes two facts of importance: first, the audiometer yielded essentially equivalent results at the beginning and at the end of the study. This finding may be accepted as evidence of the instrument's stability; second, the average thresholds yielded by normal ears were consistently close to the zero hearing loss value. One could, for many clinical uses, accept the audiometer as being in adequate calibration; however, it was felt that for the purposes of the present study, a correction should be applied whenever the mean of the combined data for normals deviated more than 2.5 db from the zero loss value. In consequence, all references to the audiometric results obtained in this study are hereafter reported in terms of these corrections, which involved subtracting 5 db from all bone conduction readings for 250 c.p.s. and adding 5 db to all bone readings at 1,500, 2,000, 3,000 and 4,000 c.p.s. Likewise, 5 db was added to all air readings at 1,500, 2,000, 4,000 and 8,000 c.p.s.

Additional checks were made on the corrected calibration of the bone conduction system. For one thing, both air and bone tests were administered to 12 ears having perceptive loss, following the theory that a well calibrated audiometer

should yield identical air and bone thresholds when the subject has a pure "inner ear" impairment. Fig. 3 shows that the averages of the corrected air and corrected bone thresholds yielded by these 12 ears agreed closely with each other.

Second, the same comparison was made with normal ears. Fig. 4 summarizes the agreement between the two sets of corrected thresholds which emerged when 10 normal hearers were each tested twice. The equivalence was even better for the normals, but the tendency observed with perceptive cases for bone readings to be slightly better than air was reversed for the normals.* It is noteworthy that, when the two groups of data were combined as shown in Fig. 5, the average thresholds for air are in almost perfect agreement with those for bone. It is apparent that the corrected calibrations of the two systems were highly equivalent. This conclusion was confirmed by the further discovery that the Maico E-2 gave results which were in close agreement with those obtained from a second audiometer whose calibration had been demonstrated clinically to be particularly good. Thus, the calibration of the pure tone audiometer used in the present study was judged to be highly adequate for the purposes at hand.

In order to determine whether the performance of the audiometer remained constant throughout the experiment, an artificial ear was used daily to measure its acoustic output.† Table 1 shows that over a period of seven months, the acoustic output of the pure tone audiometer remained highly stable.‡ At six of the nine test frequencies, the range from the highest to the lowest reading over the entire seven months' period was 2 db or less. The greatest range was only 2.6 db.

The stability of the speech audiometer was also determined with the artificial ear, the 1,000 c.p.s. calibration tone on the

*This trend may have been due in part to masking from residual room noise, even though all tests on normals were made in the soundproof chamber in the Northwestern University Audiological Laboratories.

†The artificial ear and its amplifier were manufactured by the Audio Development Co. It included a 9-A coupler, a 640-AA Western Electric microphone, and a cathode follower which fed into a vacuum tube voltmeter.

‡Appropriate equipment for similar measurements of bone conduction output was not available, but it is believed that the bone system was also satisfactorily stable. The reason for this opinion is that normal ears, as already reported, yielded essentially the same average bone thresholds at the beginning and at the end of the experiment.

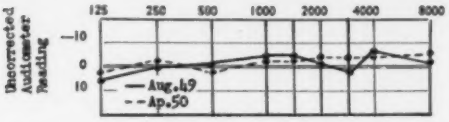

Fig. 1. Averages of air conduction thresholds yielded by 10 subjects with normal hearing.

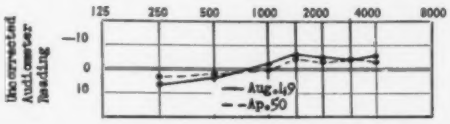

Fig. 2. Averages of bone conduction thresholds yielded by 10 subjects with normal hearing.

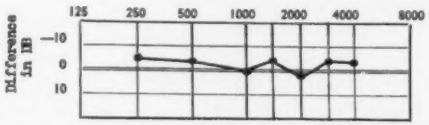

Fig. 3. Agreement between averages of corrected air and bone thresholds yielded by 12 patients with pure nerve losses. Values below zero line indicate that bone was better than air.

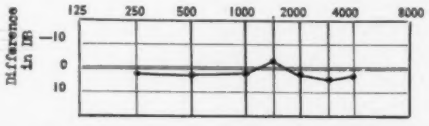

Fig. 4. Agreement between averages of corrected air and bone thresholds yielded by 10 subjects with normal hearing. Values above zero line indicate that bone was better than air.

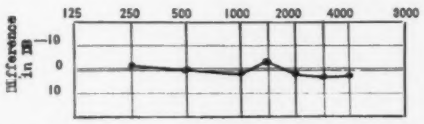

Fig. 5. Agreement between averages of corrected air and bone thresholds for combined group of 10 subjects with normal hearing and 12 patients with pure nerve losses. Values above zero line indicate that bone was better than air.

TABLE 1.

Summary of Daily Measurements on Acoustic Output at Arbitrary Settings of the Maico E-2 Audiometer.
Results Expressed in Decibels re .0002 Dynes/Cm².

Frequency	Mean SPL	Lowest Reading	Highest Reading	Range from Lowest to Highest
125	104.0	102.6	105.2	2.6
250	104.9	103.9	105.5	1.6
500	104.4	103.5	105.1	1.6
1,000	102.7	101.8	103.4	1.6
1,500	101.3	100.3	102.3	2.0
2,000	101.2	99.8	101.8	2.0
3,000	99.9	98.6	100.4	1.8
4,000	103.6	102.0	104.6	2.6
8,000	101.0	99.7	101.9	2.2

test record being used as the acoustic signal during the daily measure on output. The total range of all readings during the entire experiment was only 1.8 db. The adequacy of calibration for this audiometer was checked empirically by obtaining the speech thresholds of normal ears at intervals throughout the study.

The foregoing measurements gave the writers confidence that the test instruments were in satisfactory calibration and that they possessed high consistency in output throughout the time span of the study.

— IV —

As would be expected in any consecutive series of patients selected for the fenestration operation, the 58 cases under investigation varied substantially in the preoperative levels of their hearing. The average preoperative acuity for the entire group is reported in Fig. 6.

The mean impairment by air for pure tones in the ears selected for surgery varied from one frequency to another and ranged between 55 and 60 db. The average losses in the opposite ear were slightly smaller, lying between 45 and 50 db. The ears selected for surgery showed an average loss for speech of 57 db, while the contralateral ears averaged 43 db

of loss for speech. These discrepancies between ears reflect the surgeons' practice of choosing the poorer ear for operation.

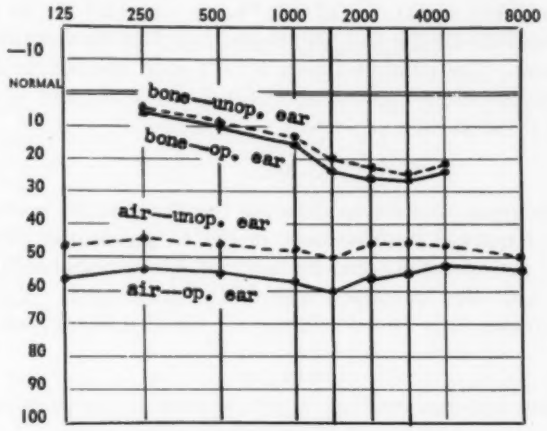

Fig. 6. Average preoperative hearing of 58 patients selected for fenestration surgery.

By contrast, as is also evident in Fig. 6, the bone conduction acuity of the ears scheduled for surgery was only slightly inferior to the acuity for the opposite ears. The separation between the mean values for the two ears was not more than 3.3 db for any test frequency. The two curves slope off relatively uniformly, with the least loss being approximately 5 db for 250 c.p.s. A slight depression may be noted in the region from 1,500 through 3,000 c.p.s., so that the total configuration resembles the profile of a skillet. Considering the overall picture, the average levels were near normal — as would be expected in a population selected for fenestration surgery.

The mean age for the group was 39 years. Two-thirds were between 29.5 and 48.5, while the youngest was 19 and the oldest was 60. Half of the subjects were under 38 years old.

Viewed in terms of Shambaugh's categories of suitability for surgery,[6] the group consisted of 31 class "A," 25 "B,"

and two "C" cases. In the age group under 38 there were 19 "A" and 10 "B" cases, while those over 38 included 12 "A," 15 "B," and two "C" cases. Considering only the ears selected for surgery, the average bone loss in the speech frequencies (500 to 2,000 c.p.s.) for all "A" cases was 12.8 db, as compared to 23.0 db for the "B" cases, and 34.4 db for the two "C" cases. The average losses by air over the 500 to 2,000 c.p.s. range were, respectively, 52.0 db, 59.2 db, and 62.5 db for the three groups.

— V —

In the group under consideration the fenestration operation produced marked improvement in the air conduction thresholds for the operated ears. By contrast, the thresholds for the unoperated ears remained essentially unchanged. Table 2 demonstrates this fact in terms of spondee thresholds and

TABLE 2.

Comparison of Mean Preoperative and Postoperative Levels of Losses for Air Conduction Stimuli.

	Operated Ears			Unoperated Ears		
	Preop. Level	Postop. Level	Gain in Db	Preop. Level	Postop. Level	Gain in Db
a. — Average of 500, 1,000, 1,500 and 2,000 c.p.s.						
Under 38	53.7	28.5	25.2	44.1	42.4	1.7
Over 38	60.2	33.9	26.3	51.4	50.8	0.6
Total Group	57.0	31.1	25.9	47.8	46.6	1.2
b. — Spondee Threshold.						
Under 38	53.4	28.2	25.2	44.1	41.6	2.5
Over 38	60.5	33.3	27.2	52.2	50.2	2.0
Total Group	57.0	30.8	26.2	48.1	45.9	2.2

average pure tone hearing in the speech range. Figs. 7 and 8 supply the analogous information for the various test tones in the audiometric range.

Since the mean change in the unoperated ears was very small, almost the total gain in the operated ears may be accepted as surgical improvement. The stability of unoperated ears allows a high degree of confidence in the audiometric results for the operated ears. The average improvement

which the latter achieved at each test frequency is, therefore, worthy of review. Table 3 reports the pertinent statistics, which may be summarized as follows:

TABLE 3.

Summary by Frequencies of Average Gain in Air Conduction Acuity Produced by Fenestration Surgery.

Test Frequency	Mean Improvement in Decibels	Standard Deviation of Improvement	Standard Error of Mean
125	23.5	11.5	1.5
250	24.4	10.7	1.4
500	25.1	8.9	1.2
1,000	25.9	9.8	1.3
1,500	26.7	10.4	1.4
2,000	25.4	9.2	1.2
3,000	16.9	10.7	1.4
4,000	12.5	11.3	1.5
8,000	8.8	13.4	1.9

1. The average air conduction gain for frequencies from 125 c.p.s through 2,000 c.p.s was approximately 25 db.

2. The average air conduction gain became progressively less above 2,000 c.p.s., being approximately 17 db at 3,000 c.p.s., 13 db at 4,000 c.p.s., and 9 db at 8,000 c.p.s. This tendency is in general agreement with the findings of Thurlow, Davis and their associates.[5]

Furthermore, on the basis of *T* tests, comparing the gains at different frequencies, the configuration as described may be accepted as a valid representation of the group's average improvement by air when measured three months postoperatively.

When coefficients of correlation were computed, a fairly high interdependence was found to exist between the air conduction gains for adjacent frequencies. The values of *r* ranged from 0.54 to 0.73. The parallelism was slightly more pronounced for tones in the low frequency range. Individual variations existed, but the improvement which a patient received tended to be generalized and to reveal itself proportionately over a wide frequency range.

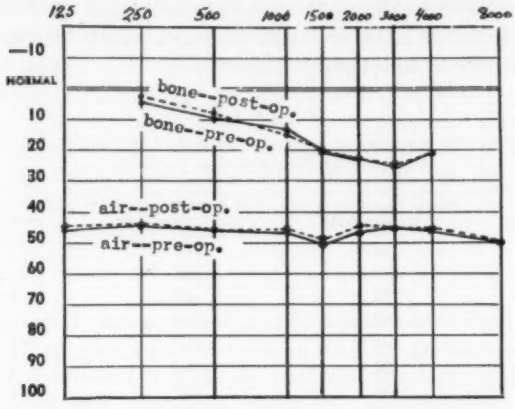

Fig. 7. Comparison between average preoperative and average postoperative thresholds for air and for bone obtained from the unoperated ears of 58 patients who underwent fenestration surgery.

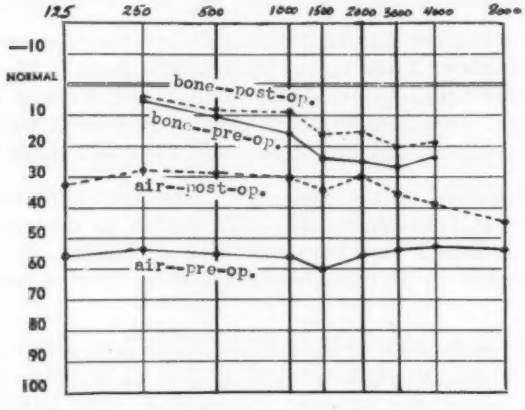

Fig. 8. Comparison between the average preoperative and average postoperative thresholds for air and for bone obtained from 58 ears subjected to fenestration surgery.

There was a substantial tendency for the patients with greatest losses to achieve greatest postoperative gains; however, statistical analysis showed that the total loss *per se*

was not the factor related to gain. Instead, as would be expected, the average gain bore good interdependence with the preoperative conductive component as gauged by the air-bone gap ($R = 0.48$); however, it was apparent that the relationships were complex and that other variables were active to produce individual differences.

Age was not a critical factor in the amount of improvement in air conduction hearing, as can be seen from Table 2. The average gains for the two age groups were essentially equivalent. Older subjects showed an average postoperative loss greater than the younger subjects because their preoperative hearing was also poorer; therefore, to the extent that older patients tended to show greater initial impairments because of greater secondary nerve involvements, age was an indirect determinant of postoperative levels, but not a determinant of amount of improvement.

When subjects were subdivided according to suitability for surgery, the same relationship was observed. The 30 class "A" cases gained 26.7 db in the speech range, which was only slightly greater than the 24.3 db gain of 25 class "B" cases.

The "A" cases achieved a mean postoperative level of 26.1 db, while all 45 cases whose initial losses in the speech range were 60 db or less averaged 28.6 db of loss postoperatively. The acuity of the majority of the subjects was restored to a "practical level" of 30 db or better.

As shown in Table 2, improvement in spondee thresholds paralleled closely pure tone results for the 500 to 2,000 c.p.s. range. A coefficient of correlation of $r = 0.84$ was found between the gain as expressed by the 500 to 2,000 c.p.s average and the gain as measured with spondees. The conclusion which emerges from these findings is that, insofar as the present subjects are typical, otosclerosis produces a relatively uncomplicated impairment in the sense that acuity for pure tones and acuity for speech are not reduced disproportionately to one another. The two modalities show a similar parallelism when postsurgical results are considered. Both offer good estimates either of preoperative hearing loss or of surgical

benefit. This conclusion is somewhat different from the interpretation made by Davis and his associates,[8] but the disagreement is amply explained by the fact that these investigators included data for 2,896 c.p.s in their computations.

— VI —

In the group under consideration, the fenestration operation produced a definite shift in the bone conduction thresholds for the operated ear, while thresholds in the unoperated

TABLE 4.

Comparison of Mean Preoperative and Postoperative Bone Conduction Levels Averaged Across the 500 to 2,000 C.P.S. Range.

	Operated Ear			Unoperated Ear		
	Preop. Level	Postop. Level	Gain in Db	Preop. Level	Postop. Level	Gain in Db
Under 38	14.9	9.5	5.4	13.8	13.2	0.6
Over 38	21.7	15.3	6.4	19.0	19.6	0.6
Combined	18.3	12.4	5.9	16.4	16.4	0.0

ears remained essentially unchanged. Table 4 demonstrates this fact for the 500 to 2,000 c.p.s. averages. Figs. 7 and 8 summarize the results by individual frequencies when average thresholds are plotted on the audiogram. Fig. 9 gives a more precise picture of the average shifts observed.

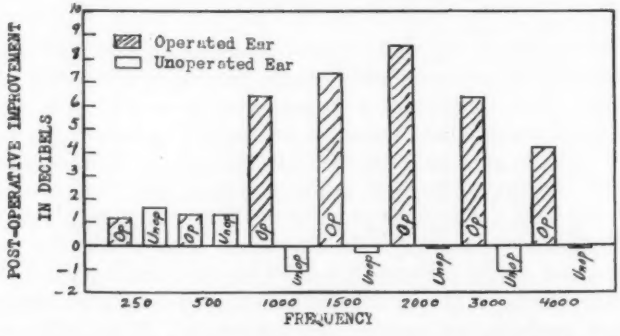

Fig. 9. Comparison between mean improvements in bone conduction sensitivity for operated and for unoperated ears of 58 patients.

Nonfenestrated ears revealed virtually no shift, inasmuch as the variation between pre- and postoperative bone thresholds was at no frequency greater than 1.6 db. By contrast, postoperative averages for fenestrated ears revealed gains over preoperative levels of 1.2 db at 250 c.p.s., 1.4 db at 500 c.p.s., 6.5 db at 1,000 c.p.s., 7.4 db at 1,500 c.p.s., 8.5 db at 2,000 c.p.s., 6.4 db at 3,000 c.p.s., and 4.2 db at 4,000 c.p.s. Thus, the operated ears revealed a group pattern in which almost no shift occurred for test tones below 1,000 c.p.s., but a fairly uniform shift averaging 6.6 db occurred throughout the range from 1,000 c.p.s. through 4,000 c.p.s.; furthermore, when the data were subjected to statistical tests of significance, it became clear that the small shifts evidenced by unoperated ears could be attributed to chance, while the larger changes in bone sensitivity found from 1,000 c.p.s. through 4,000 c.p.s. in the operated ears were attributable to surgery. The foregoing results are important in demonstrating two things: First, since fenestration surgery ordinarily modifies only the conductive component of the hearing loss, mechanical factors may modify bone conduction thresholds to a measurable degree. Second, fenestration surgery produces a mechanical shift in response to bone stimuli which is a partial reverse of the preoperative otosclerotic notch in the bone curve described by Carhart.[1]

Individual patients deviated from the foregoing pattern to a substantial degree, so that this pattern must be viewed as only a group trend. For example, the average bone gain (over the 500 to 2,000 c.p.s band) for class "A" cases was 5.5 db, while it was 6.4 db for class "B" cases, and 7.5 db for the two class "C" cases; however, although the correlation was low ($R = 0.35 \pm 0.11$), a slight tendency was noted for greater gains to be associated with greatest initial losses for bone. In contrast, within the limits represented by the patients subjected to study, the amount of bone gain was not found to be a function of the preoperative magnitude of the conductive component of loss as gauged by the preoperative air-bone gap ($r = -0.26 \pm 0.12$), nor was it a function of the age of the patient. Finally, postoperative thresholds for bone at adja-

cent test frequencies showed a moderate tendency toward parallel shifts at the extremes of the audiometric range, but the parallelism was substantially reduced for central frequencies. Marked individual differences existed in the pattern of postoperative gain for bone.

The only conclusion which appears possible from the configuration of results just outlined is that factors as yet unknown were at work in individual cases. When these factors are discovered through future research they may help us to understand and to evaluate individual cases. In the meantime, we may accept the fact that a clear pattern of bone conduction gain is evident as a group trend.

— VII —

Even though the mechanical factors that influence bone conduction thresholds of otosclerotics are such that the bone audiogram gives only an approximate picture of cochlear reserve, it is, nonetheless, interesting to review the effect of fenestration surgery upon the gap between the air and bone curves.

Air-bone gaps, for the ears which underwent surgery, were computed for the 500 to 2,000 c.p.s. range. The average initial air-bone gap for all 58 subjects was 38.7 db and the mean postoperative gap was 18.8 db. Preoperative gaps averaged 40.0 db for the 30 class "A" cases. Their residual gaps averaged 18.8 db after surgery, so that the mean reduction in the size of the gaps was 21.2 db. The initial gaps for 25 class "B" cases averaged 36.2 db, while their average reduction was 17.9 db and their residual level was 18.3 db. Thus, class "B" was left with a postoperative mean which agreed almost identically with that of the class "A" cases.

The implication from the foregoing figures is obvious: namely, the residue of conductive component following surgery was, on the average, independent of the magnitude of original impairment. Individuals deviated from the group average, yet the operation, when viewed in terms of only the conductive component, tended to bring all patients to the

same terminal point, regardless of preoperative differentials. This finding confirms the Davis and Walsh concept of a postoperative conductive impairment which is unavoidable because the ossicular mechanism is disrupted surgically.[2]

The size of the gap differed from one frequency to another. Table 5 summarizes results for the group as a whole. The following trends are apparent from the table: First, the initial air-bone gap showed a progressive decrease with rise in the test frequency, being 47.6 db at 250 c.p.s., but only 26.5 db and 29.1 db for 3,000 and 4,000 c.p.s., respectively. Second, the mean reduction in gap resulting from surgery was progressively less with increased frequency. Last, the residual air-bone gap was smallest for the middle frequencies, being 14.3 db at 2,000 c.p.s. In contrast, the residual gaps for 250, 500, 1,000 and 4,000 c.p.s. were all greater than 20 db. The possible significance of those variations is discussed below. For the moment, the point to emphasize is that the conductive component of the loss due to clinical otosclerosis is not equally modified at different frequencies by surgery.

TABLE 5.

Comparison of Initial and Residual Air-bone Gaps at Individual Frequencies.

Test Frequency	Mean Initial Gap in Db	Mean Residual Gap in Db	Mean Reduction
250	47.6	24.4	23.2
500	44.7	20.8	23.9
1,000	40.7	21.2	19.5
1,500	37.6	18.3	19.3
2,000	31.2	14.3	16.9
3,000	26.5	15.9	10.6
4,000	29.5	20.8	8.3

— VIII —

The findings reviewed above demonstrate clearly that surgical reorganizaton of the middle ear structures does change bone conduction thresholds. One immediately wonders whether the postoperative bone thresholds are true indications of

cochlear sensitivity or whether they, too, are displaced by residual mechanical influences. In other words, one wonders whether the postoperative bone audiogram gives an undistorted picture of cochlear function or whether the picture is obscured by variations in response due to the physical state of the surgically modified ear.

The data of the present study were analyzed from the standpoint of the foregoing question. Basically, the question may be phrased as follows: "Is the postoperative separation between a patient's air and bone conduction thresholds equal to the residual conductive impairment he possesses?" If so, his bone audiogram is determined fully by his cochlear reserve, and, if not, his bone audiogram must still incorporate an additional mechanical effect. Analysis of this question consisted of determining in the manner described below how well the air-bone gaps observed after surgery agreed with the patient's residual conductive impairment as computed by an alternative method.

The first step was to estimate residual conductive impairment without relying upon bone thresholds in doing so. Davis and Walsh have demonstrated that such an estimate may be derived entirely from the air conduction thresholds yielded by groups of patients subjected to fenestration surgery.[2] Their procedure, which they employ with speech thresholds, is simple. It is illustrated in Fig. 10 with the speech thresholds obtained in our own study. The preoperative and postoperative results for fenestrated ears are plotted against one another. It will be observed that the results are not randomly distributed. Instead, they form a scattergram with a definite pattern which allows one to specify the limit of surgical restoration of hearing.

It can be seen in Fig. 10 that a number of patients with preoperative losses of 60 db or less had their hearing restored sufficiently so that their residual losses were about 20 db, but only a few achieved results which were even slightly better than this. Thus the group evidenced a limit in surgical benefit. Davis and Walsh assume that this limit, which their data

also show to be 20 db, is the inevitable price of disrupting the ossicular mechanism through the fenestration operation. In other words, the operation leaves a residual conductive impairment whose magnitude may be estimated as equalling the deficit in acuity which the superior surgical result retains. This residual conductive impairment is represented in Fig. 10 by the vertical portion of the boundary designated as the Limit of Anticipated Restoration.

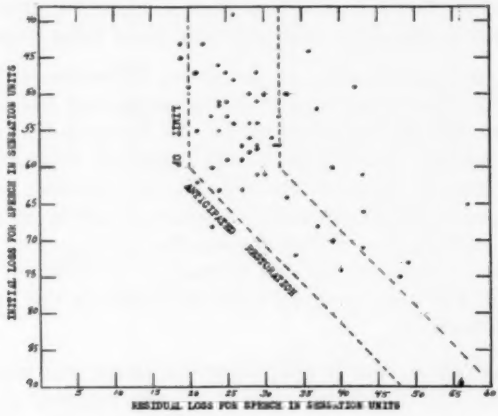

Fig. 10. Distribution of speech thresholds showing surgical results in relation to preoperative hearing level.

Obviously, a patient with a nerve loss will fall short of restoration to normal hearing by an amount at least equal to the aforementioned conductive residue plus his degree of nerve loss. The sloping section of the Limit of Anticipated Restoration shown in Fig. 10 represents this fact. It indicates that the cases with *more severe* preoperative losses failed to reach the 20 db residue. Those in this latter category who obtained the best surgical results had postoperative losses which exceeded 20 db by an amount equalling the difference between their preoperative losses and 60 db.

Stated in another way, when results are plotted as shown in Fig. 10 and a boundary is arbitrarily drawn so that it encom-

passes the best results for the group, one may estimate the maximum preoperative conductive loss produced by stapes fixation. This maximum is indicated by the point at which the boundary breaks from its vertical course and begins to diagonal to the right. In the present instance, as in Davis' and Walsh's study, this break is associated with a preoperative loss of 60 db. One concludes that the maximum loss for speech due to the conductive lesion of stapes fixation did not exceed 60 db. Patients with greater impairment than this possessed nerve involvement in addition to a conductive involvement, as, of course, did some patients with lesser total impairment.

The fundamental point in the above discussion is that two important values may be derived by employing the foregoing procedure and by disregarding for the moment the bone conduction results. One value is the maximum amount of conductive loss to be expected from stapes fixation, while the other value is the postoperative conductive deficit produced by surgical interference with the ossicular system. The latter is our primary concern, but the former is of sufficient interest so that it will also be given brief attention in the discussion which follows.

An analysis similar to that described above was made of the air conduction thresholds yielded by our subjects at each of the audiometric frequencies we employed. Fig. 11, which summarizes the data for 2,000 c.p.s., is typical of the results that emerged. Here, an Estimated Limit of Operative Restoration was arbitrarily drawn so that few scores lay to the left of it, but so that the majority lay along the boundary or bunched close to it on the right. Assuming the principles postulated by Davis and Walsh, the vertical portion of the boundary indicates the approximate size of the conductive deficit due to ossicular disruption. This vertical portion is, in the present instance, 20 db to the right of zero on the postoperative scale. One, therefore, estimates that the minimal conductive deficit to be expected postoperatively at 2,000 c.p.s. is 20 db. Moreover, the point on the preoperative scale at which the boundary breaks to take a diagonal course is an indication of the maximal conductive impairment which stapes fixation pro-

duces preoperatively at the frequency under study. The present data led to the belief that this maximum is 55 db for 2,000 c.p.s.

Similar analysis was made of the scores for each frequency from 125 through 8,000 c.p.s. The findings are summarized in Fig. 12, which is in the form of an audiogram.

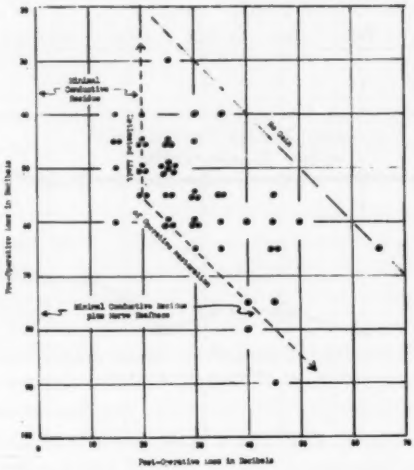

Fig. 11. Distribution of air conduction thresholds for 2,000 c.p.s. showing surgical results in relation to preoperative hearing level.

The lower curve of Fig. 12 plots the maximal conductive impairment to be expected preoperatively at each frequency. It is interesting to note that the average of the maxima across the 250 to 2,000 c.p.s. range is about 60 db* and that the maxima are somewhat smaller than this for still higher frequencies.

Our main interest at the moment is the size of the postoperative conductive deficits associated with different test

*Occasional clinical cases are encountered wherein the maximal conductive involvement at low frequencies appears to exceed 60 db appreciably, but these cases are sufficiently rare so that they do not exert an important influence on the results obtained in analyzing group trends.

frequencies. The latter are shown by the upper curve in Fig. 12. The reader will observe that these deficits appear to be constant from one pure tone to another. The findings are interpreted to mean that, as judged from the results of the 58 fenestration cases under consideration, the minimal conductive deficit following ossicular disruption was approximately 20 db across the frequency range from 125 through 4,000 c.p.s. and 15 db at 8,000 c.p.s.

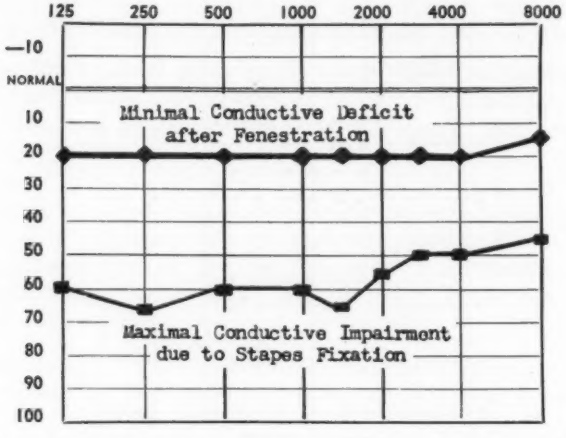

Fig. 12. Estimated limiting values of conductive impairment found in otosclerosis.

— IX —

It is the foregoing estimate, derived entirely from air conduction measurements, which may be compared with the average air-bone gaps for the same group of subjects in order to judge whether or not their postoperative bone thresholds were undistorted indications of cochlear function. As will be recalled, the size of the postoperative air-bone gaps varied from one frequency to another across the band from 250 through 4,000 c.p.s., which was the range employed for bone measurements in the present study. Now, remembering that the actual conductive deficit was estimated to be about 20 db for these frequencies, the amount by which the air-bone gap

at any frequency deviated from 20 db may be interpreted as indicating the mechanical displacement in bone response which persisted at that frequency after surgery. Pursuing the foregoing assumption, one needs merely to compute the differences involved in order to estimate the effect of mechanical influences on the postsurgical audiogram. These differences, as derived from the present study, were: –4.4 db at 250 c.p.s., –0.8 at 500, –1.2 at 1,000, 1.7 at 1,500, 5.6 at 2,000, 4.1 at 3,000, and –0.8 at 4,000 c.p.s. These values appear to be a reasonable indication, from the data at hand, of the amount by which the bone response remained mechanically modified after surgery. One may hypothecate that, on the average, bone thresholds obtained after surgery failed by the amounts listed above to give a true picture of cochlear function.

— X —

Mention has been made of Carhart's observation that a typical distortion of the bone conduction audiogram appears preoperatively as a result of the mechanical influences exerted by stapes fixation. The fact that the fenestration operation changes response to bone conduction stimuli may be viewed as indirect evidence that such distortion of the audiogram existed prior to surgery; however, there remains the problem of specifying the pattern and magnitude of the preoperative distortion in the bone curve. Without such information, estimates of cochlear function derived from preoperative bone audiograms must lack the precision one would like.

It is a simple matter to compute, for the group under study and from the information at hand, the approximate size of the average preoperative displacement of the bone curve due to the mechanical influences of stapes fixation. The procedure is shown graphically in Fig. 13. One first draws Curve A, which summarizes the gains in bone sensitivity actually produced by surgery. These gains have already been reported in Fig. 9; however, in the present instance the curve is drawn below the zero reference line because we are at the moment imagining a preoperative condition; that is, the fraction of the mechanical depression which subsequent surgery was ob-

served to reverse. One next draws Curve B, which is the mechanical distortion that we have just estimated to have remained after surgery. This curve represents the fraction of the original distortion which, according to our estimate, surgery did not eliminate. Finally, Curves A and B are com-

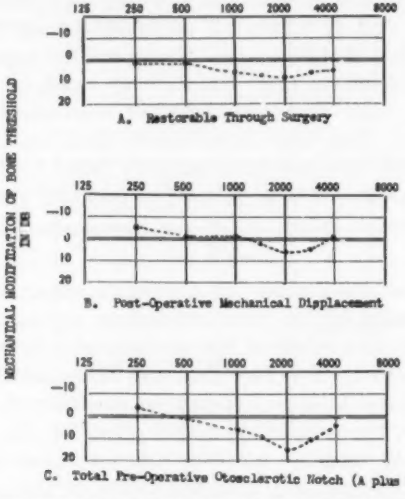

Fig. 13. Calculation of the magnitude of the otosclerotic notch (Curve C). Derived by combining the bone sensitivity restorable through surgery (Curve A) with the postoperative residue of mechanical displacement (Curve B), which was estimated from the auditory responses of 58 patients.

bined to yield Curve C, which is our computation of the size of the preoperative distortion in bone sensitivity due to stapes fixation, *i.e.*, the size of the "otosclerotic notch." In other words, since Curve C is the sum of the observed restoration plus the postoperative residue which was not restored, it is an estimate of the average amount by which the bone measurements on the 58 subjects under consideration gave a false picture of cochlear function prior to surgery.

Expressed numerically, such a procedure gives the following values for the preoperative "otosclerotic notch," when these values are computed from the data of the present study.

Stapes fixation appears to have improved bone sensitivity 3.2 db at 250 c.p.s., while mechanically depressing bone sensitivity, 0.6 db at 500, 5.3 at 1,000, 14.1 at 2,000, and 3.4 db at 4,000 c.p.s. It is interesting that Carhart,[1] in his original rough description of notch size, postulated no shift at 250 c.p.s., 5 db depression at 500, 10 at 1,000, 15 at 2,000, and 5 db at 4,000 c.p.s. The discrepancies between the present estimate and Carhart's original figures are not large. The data at hand leads to the belief that the original postulation was not seriously in error and that, on the average, the "otosclerotic notch" may be assumed to have approximately the shape heretofore described. There will, of course, be individual variations in clinical records, and the significance of these variations needs further study; however, when one remembers that the audiometer measures hearing loss in 5 db steps and that a patient will respond to the first signal stronger than his threshold, a person having an "otosclerotic notch" which corresponds to our new estimate will yield a bone audiogram which is identical with Carhart's original postulation.* It may be concluded that we have at our disposal an average set of values which apply reasonably well for most cases of clinical otosclerosis. For the moment, the values suggested by Carhart may continue to be accepted as practical ones when correcting the bone curves of individual clinical patients.

The fundamental point underlying this whole discussion is that *allowance must be made for the fact that the bone conduction audiogram gives a distorted measure of the cochlear function possessed by a patient with stapes fixation.* Thresholds obtained before operation incorporate the "otosclerotic notch." The present study demonstrates that fenestration produces a partial reversal of the notch. The study also leads to the belief that thresholds obtained after operation incorporate a lesser mechanical displacement, so that even here the bone measurements are not fully accurate indications of neurosensory function; however, appropriate allowances can

*For example, if a patient's bone sensitivity at 250 c.p.s. is —3.2 db re normal threshold, he will respond when the signal strength is zero rather than —5. Thus his audiogram will not reveal the mechanical shift in bone sensitivity which he actually possesses. Similarly, the 3.4 db depression in sensitivity at 4,000 c.p.s will be measured audiometrically as a threshold of +5 db. Analogous relationships exist at the other test frequencies.

be made when interpreting either the preoperative or the postoperative bone audiograms.* Improved estimates of cochlear reserve should usually thus be obtained. Among other things, such estimates based on postoperative tests administered at appropriate intervals may be compared with the estimate derived from the preoperative records. The stability of a patient's cochlear function may thus be judged with greater accuracy and long term changes in hearing may be followed with greater confidence.

— XI —

Since mechanical shifts in bone sensitivity are an integral part of the otosclerotic's auditory configuration, we are in a position to apply a new level of interpretation to the audiometric records of such cases. Enough has been learned to help the otologist in making fuller use of bone conduction measurements in selecting candidates for fenestration and in evaluating the results of surgery. Naturally, much more research is needed to reveal both the full nature of the phenomena involved and the variations for which allowance must be made. For example, the present study gives a description of surgical results based on tests made three months after surgery. This time lapse was probably sufficient so that hearing levels had achieved postoperative stability, but the matter needs further exploration. Nelsson[5] has demonstrated, with measurements obtained during surgery, that fenestration produces responsiveness to bone stimuli; however, the values he obtained for this improvement differ substantially from those reported in the present study. Additional research is obviously needed to plot the progression of change in bone conduction sensitivity associated with the postoperative recovery period. Recognizing our need for additional knowledge, there are, nonetheless, several conclusions which may be drawn from the present study. These conclusions are:

*For example, Shambaugh[7] has suggested using corrected bone values as the basis for predicting the anticipated air conduction audiogram following surgery. He merely adds 25 db of impairment to the corrected configuration of the preoperative bone curve. He has demonstrated that, on the whole, his predictions are usefully close to the actual surgical results which he achieves.

1. Fenestration surgery produces real improvement in responsiveness to bone stimuli throughout the range from 1,000 through 4,000 c.p.s. This fact is evidence that mechanical changes in the middle ear system modify bone conduction thresholds.

2. The postoperative residue of conductive impairment appears to have a limiting minimal value of 20 db across most of the audiometric range. Since the average air-bone gap after surgery varies from one frequency to another and fails to conform to this value, a mechanical distortion of the bone audiogram remains after surgery.

3. The magnitude of the average influence which stapes fixation has upon bone conduction thresholds may be estimated by adding the observed shift in bone sensitivity which surgery produces to the estimate of the unrestored residue of mechanical distortion. This procedure yields values which agree reasonably well with those postulated by Carhart's earlier description of the otosclerotic notch. The results thus increase the confidence with which one may state that stapes fixation tends to produce a mechanically induced distortion of the bone conduction audiogram which tends to have a typical configuration.

4. The foregoing conclusions refer to group trends. Individual patients often show substantial deviations from these trends. It is apparent that factors as yet unknown are operative and that further research directed at isolating these factors is needed.

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FUNCTIONAL STUDIES ON LABYRINTHINE FENESTRATION IN ANIMALS — II.*

MARGARET R. BENEDICT, Ph.D.,†
New York, N. Y.

I — INTRODUCTION.

Since 1942 a series of experiments designed to determine the effects of the fenestration operation upon the hearing function of animals has been performed at the University of Rochester. A first report of this research[12] described experiments in which the animals' hearing was tested by means of cochlear potentials. The present report is concerned with experiments in which the hearing of monkeys was measured by a conditioned-response technique (a method of behavioral observation) similar to that described by Culler *et al.*,[2] both before and after the fenestration operation. In addition, the middle and inner ears were examined microscopically after the postoperative hearing measurements had been completed. The results of the present series of experiments are not nearly so definitive as those of the earlier-reported experiments.[12] It is considered advisable, however, to publish them at this time because they do not agree with the conclusions which have been drawn from most of the published clinical data concerning fenestration.

Case histories of fenestrated humans have emphasized a positive relationship between patency of the fistula and hear-

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*Communication from the former Institute of Audition at the University of Rochester, Elmer A. Culler and Edward H. Kemp, Directors.

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The author makes grateful acknowledgment to Karl Lowy, M.D., who supervised the entire experiment, performed the surgery, and examined the ear sections. The author is further indebted to Prof. Alice Van Krevelen for the histological processing, and to Prof. G. R. Wendt for editorial assistance.

†Barnard College, Columbia University.

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ing improvement. It is claimed that, when an initial postoperative hearing improvement is subsequently lost, there is concomitant absence of a positive fistula symptom and (in cases where a revision was carried out) observable closure of the fistula with newly-formed bone.[3] For this reason, most previous animal research dealing with the fenestration operation has been directed toward improving surgical techniques for creating a fistula which would not close with regenerating bone.[10,11] The present research, however, was directed ultimately toward finding a correlation between anatomical structure and hearing function, and it has indicated that the postoperative hearing improvement was relatively independent of the open or closed condition of the fistula; furthermore, the results did not show any other single anatomical difference between the ears of those monkeys whose hearing was greatly improved and those whose hearing was improved little or not at all.

These rather inconclusive results would appear to be applicable primarily to the study of the mechanics of the auditory transmission system and the changes induced in this system by the fenestration operation. Although a hearing loss comparable to that found in cases of clinical otosclerosis was produced in the monkeys, the oval window was not occluded, and the fenestration operation consequently added a third functional window to the inner ear. The fact that the making of this third fenestra by surgical means appeared to produce improved hearing in some monkeys we consider an important outcome of the present study, and this report of results is made with the aim of raising interest in the problems presented.

With the purpose of obtaining the monkeys' audiograms and comparing histologic condition of the ear with them, the present experiment was divided into four fairly distinct parts:

The first part was designed to meet the problem of deafening the monkey's experimental ear in a manner as similar as possible to that of clinical otosclerosis. This was done

surgically by removing the incus to interrupt the chain of ossicles; however, since we could devise no sure method of immobilizing the oval window, it was necessary to determine the actual amount of deafness due to exclusion of the ossicular function alone. To this end, the monkeys' hearing losses following removal of the incus were determined and compared in both range and amount to the hearing loss measured in cases of clinical otosclerosis.

The second part of our experiment consisted of the fenestration operation, followed by determination of the animals' immediate postoperative changes in hearing threshold. Because the largest number of animals was used in this phase of the study, some statistics were utilized in an attempt to make some generalizations concerning the results of the operation.

The third phase was a study of the duration of the postfenestration hearing changes. The monkeys' hearing was tested at various intervals as long as their general health remained good or until some characteristic of their audiograms made histological examination of the ears desirable.

The fourth phase consisted of examining microscopically sections of the middle and inner ears and relating these observations to each animal's most recent audiogram.

II — EXPERIMENTAL PROCEDURES.

A. Animals: Because of the similarity of their ears to those of humans and because their relatively high intelligence made training them and testing their hearing fairly simple, rhesus monkeys were chosen as the experimental animals. Twenty-one monkeys, approximately equally divided as to sex and of about the same age (preadolescent) are included in the results.

B. Apparatus: The audiometric tests of the monkeys were made in a sound-insulated room similar to that described by Harris,[5] which contained a loudspeaker and the restraining cage for the animal. This room had a one-way vision window directly opposite the cage, through which the experimenter could observe the animal.

The tones presented as stimuli were generated by a General Radio Type 608-A Oscillator, amplified, attenuated in fixed steps and delivered in the test room by a dynamic loudspeaker. A brief shock stimulus of variable intensity was applied to the animal through leads to a chain harness around his chest and to the metal floor of the cage. The onset and duration of the tone and shock were controlled by means of a relay system operated by a constant-speed rotating disc.* The experimenter was able to attenuate the tone in steps of 4 db. These decibels of attenuation from a fixed sound level were recorded as the monkey's audiogram. Since all data in this study are comparisons between these audiograms, no attempt was made to translate thresholds into absolute terms.

C. Training: The monkeys were trained by *instrumental avoidance conditioning* techniques. That is, they learned that if they gave some response (for example, waving a paw) following the presentation of a tone, they would thereby avoid the shock which would otherwise follow that tone. This habit was quite easy to establish and extremely stable once it had been established. It was then generalized to include responses to both loud and soft tones with frequencies of 150, 500, 1,000 and 4,000 cycles per second.

D. Testing: Once the monkeys were thoroughly trained, it was possible to test their hearing in a manner which followed in most significant details the method described by Harris.[5] In determining the threshold for each of the four stimulus frequencies used, a modified form of the psychophysical Method of Limits — the so-called Method of Serial Groups[5]— was utilized. The threshold was defined as that intensity at which the animal made his response 50 per cent of the time. The monkey was given five trials at each intensity level and was considered to have "passed" a given level if he responded correctly in three or more of the trials. For each frequency a descending, ascending, and descending series of trials were given in that order so that the animal's threshold was crossed three times and, therefore, the determination of

*This apparatus was designed and maintained while he was at Rochester by Prof. John D. Coakley.

his absolute intensity limen was made from at least 15 individual tests of his hearing at near-liminal intensity. The monkey's audiogram for the four frequencies used was in most cases considered valid when four consecutive limens at each frequency (determined on different days) varied by no more than 4 db.

E. Surgery: The operations were carried out while the animals were under deep nembutal anesthesia, and standard aseptic technique was employed throughout. With the exception of a few of the control operations, all operations were performed by the same surgeon.* Adequate recovery time was allowed following each operation before the animals' hearing was tested; and nystagmus and indications of infection were checked postoperatively.

The experimental deafening of one of the ears was achieved by a simple mastoidectomy and removal of the incus. In the early cases the head of the malleus was removed also, but it was soon found more expedient to postpone this step until the time of the fenestration operation.

The control operation was designed to eliminate the functioning of the monkey's other ear, or, failing this, to render it at least as deaf as the experimental ear. The ossicular chain was again interrupted by removal of the incus, and 70 per cent alcohol was injected into the labyrinth through a fistula made in the horizontal semicircular canal. Histologic observations of the destruction produced in the inner ear by this procedure will be discussed later.

The fenestration operation itself followed fairly closely the technique described by Lempert,[7-9] but postauricular incisions were used exclusively. In all cases the incus was absent, and the head of the malleus was removed where it had not been removed previously. The fistula was made as far anteriorly as possible and covered with a meatal flap (in several of the operations, however, the flap was torn and replaced with a

*Dr. Karl Lowy, who has worked with Dr. Lempert, employed an operative technique almost identical with that used on his human patients. Only minor modifications (such as a postauricular incision) were necessitated by the monkey anatomy.

skin graft from the back of the auricle); also, in a few cases, a piece of cartilage was inserted into the fistula before covering it. In most animals a paraffin plug was used to hold the flap in place. In a few cases Gelfoam packing was used.

Except in the few cases where absorbable Gelfoam had been used as packing, the fenestrated ear was reoperated approximately seven to 12 days later when the packing or paraffin plug was removed. At this time the healing of the flap was noted. Since this final operation was invariably bloody, one to two weeks were allowed to elapse before testing the animal's hearing; by then it was assumed that most of the blood had been absorbed.

Recovery of the animals from these operations was remarkably rapid. Except for a few days following the control operation — where the ear had been assaulted with alcohol — during which the animals showed dizziness, nystagmus, and temporary or permanent torticollis, functional recovery of the animal coincided with recovery from the anesthetic.

F. Histology: When hearing tests had been completed for an animal, its head was perfused with formalin and the temporal bone removed bilaterally. These temporal bones were fixed in formalin, decalcified in trichloracetic acid, and embedded in celloidin. They were then sectioned at 20 $m\mu$ and mounted on slides. Finally they were stained with Masson's trichrome stain.* All of the experimental ears and a random sample of the control ears were examined microscopically.†

G. Statistics: The statistical methods for the treatment of small samples described by Fisher[4] were used in treating the data obtained. The reliability of all the obtained differences between means of the auditory limens was tested by means of "Student's t." When the probability that the two sample means being compared were drawn from the same population was greater than 0.05, that fact is indicated in our tables by

*All of the histological processing was performed by Alice Van Krevelen.

†All of the slides were examined by Dr. Lowy, who dictated the descriptions of his findings reported later in this paper.

parentheses around the reported difference. When grouping the data, these nonsignificant differences are treated as zero differences.

III — RESULTS.

A. Hearing Loss Due to Removal of the Incus: This part of the experiment was carried out upon a series of four monkeys of approximately the same age, size, health, and length of sojourn in the laboratory. The left ear of each animal was subjected to the control operation of removing the incus and injecting alcohol into the labyrinth. Then the hearing threshold of the intact right ear was determined for each of the four test frequencies. Following this preoperative determination of thresholds, the incus was removed from the right ear, and the animals' limens were determined postoperatively. The average thresholds before and after removal of the incus were calculated, and the difference between them taken as the hearing loss.

Table I shows the monaural hearing thresholds before and after removal of the incus in the experimental ear. Each threshold tabulated is an average of four or more threshold

TABLE I.

MONAURAL HEARING THRESHOLDS BEFORE AND AFTER INCUS REMOVAL.

Monkey	Frequency							
	150 cps		500 cps		1,000 cps		4,000 cps	
	before	after	before	after	before	after	before	after
Ermintrude	59.3 db	11.2 db	73.3	18.5	73.5	29.6	76.0	31.5
Agatha	63.2	11.6	70.0	19.5	74.5	30.0	74.4	31.0
Moe	57.0	14.3	68.0	27.6	73.0	36.6	74.0	29.6
Alan	47.7	7.5	59.5	17.5	70.4	26.0	67.5	25.0
Mean	56.8	11.2	67.7	20.8	72.8	30.6	72.9	29.3
Mean Loss	45.6		46.9		42.3		43.7	

determinations made on successive days within a period of two weeks or less. A mean loss — the difference between the two series of thresholds — was calculated for each frequency. It may be observed that this loss was approximately the same in magnitude for the four frequencies tested; the general average of these four was 44.6 db.

B. Changes in Hearing Immediately After the Fenestration Operation: This part of the experiment was carried out on a series of 19 monkeys over a period of five years. These animals were of assorted age, size, sex, and length of sojourn in the laboratory. With the exception of two animals who were previously used in Part I, all of the monkeys were first operated upon — the incus removed from the experimental ear, and the other ear deafened by the control operation. The animals were then conditioned and a series of limens determined. The means of these limens were taken as the monkeys' prefenestration audiogram.

At this time the fenestration operation was performed upon the experimental ear. Approximately one and one-half weeks after the ear had been reoperated to remove the packing (if nonabsorbable packing had been used), when external examination indicated healing and absorption of blood in the ear, a second set of limens was taken. The means of these limens served as the postfenestration audiogram. The differences in decibels between these two audiograms were taken as the change in the animals' hearing due to the fenestration operation.

Table II shows the amount of hearing change in decibels for each animal. In this table the monkeys are grouped according to breadth of their hearing improvement from those who showed improvement at all four frequencies to those who showed improvement at none. From this grouping it can be seen that 17, or 89 per cent, of the animals showed a significant improvement in hearing for at least one frequency. Only two of the animals who exhibited improvement at some frequencies suffered significant losses at others.

Table III shows the data, isolated from Table II, of the four monkeys who showed significant losses following the fenestration operation. These animals represent 21 per cent of the total group.

Table IV shows the results of treating the data in Table II separately for each frequency. It can be seen that the highest percentage of animals made gains at 1,000 cps and the great-

TABLE II.

CHANGES IN HEARING IMMEDIATELY FOLLOWING FENESTRATION OPERATION.

(Parentheses indicate a nonsignificant value.)

Monkey	150 cps	500 cps	1,000 cps	4,000 cps
Gains at all four frequencies N-3, 15.8%				
Bennie	9.0 db	9.4 db	7.7 db	13.0 db
Mehitabel	5.5	6.5	9.3	8.5
Karmon	8.5	9.3	10.2	8.4
Gains at three frequencies N-7, 36.8%				
Joe	6.8	7.5	4.3	(2.0)
Mrs. Adolph	11.5	17.0	8.2	(0.9)
Phineas	2.6	(1.2)	7.7	12.1
Winston	9.5	–4.5	6.5	22.5
Mabel	8.3	(0.4)	10.7	4.0
Muggsy	(2.0)	4.5	3.5	3.5
Dunois	(2.0)	7.5	6.0	5.7
Gains at two frequencies N-5, 26.3%				
Cleopatra	4.0	3.1	(–0.7)	(1.5)
Clarissa	7.6	(0.3)	(3.0)	5.3
Kan	(2.0)	4.0	8.4	(2.6)
Angelpuss	–6.7	3.7	–3.3	8.7
Bertram	(–1.5)	(–8.4)	10.3	8.9
Gains at one frequency N-2, 10.5%				
Kopeck	4.0	(–2.9)	(–1.5)	(–0.4)
Ermintrude	(–0.2)	(0.2)	10.4	(0.5)
No improvement N-2, 10.5%				
Alma	–3.6	0	(3.0)	(1.7)
Agatha	–5.2	(4.2)	0	(0.7)

TABLE III.

SIGNIFICANT LOSSES IN HEARING FOLLOWING FENESTRATION.

Monkey	150 cps	500 cps	1,000 cps	4,000 cps
Alma	–3.6 db			
Agatha	–5.2			
Angelpuss	–6.7		–3.3	
Winston		–4.5		

est average gain was recorded at 4,000 cps. Since the initial losses at these two higher frequencies were slightly less than

TABLE IV.

STATISTICS FOR THE GROUPS OF ANIMALS SHOWING IMPROVEMENT AT EACH FREQUENCY.

	150 cps	500 cps	1,000 cps	4,000 cps
Number of monkeys gaining	11	10	13	11
% of total N (19)	57.9%	52.6%	68.4%	57.9%
Average gain	7.0 db	7.2 db	7.9 db	9.1 db
Average loss (from Results A)	45.6 db	47.0 db	42.3 db	43.7 db
% of loss restored	15.4%	15.3%	18.7%	20.8%

those at the lower frequencies, the percentage of hearing restored by the operation was greatest for the two higher frequencies.

C. Duration of Postoperative Hearing Changes: Selection of the animals for this part of the experiment was determined primarily by the animals' health. In the early design of the experiment it was assumed that the monkeys would be alive so that their hearing might be tested one year after the fenestration; however, only five of the early animals survived until this test could be made.* This left many monkeys whose ears were histologically processed after several months had elapsed since their last hearing test. To obviate lack of data on hearing just prior to death, periodic testing of the later animals was performed at intervals of two to three months. Six animals exhibiting fairly wide variation of immediate postoperative hearing change were available for this periodic retesting.

The fluctuations in the monkeys' hearing over the period during which they were being tested produced some interesting data. Instead of the expected random variations with a tendency toward leveling off, some of the animals showed distinct trends of hearing improvement at some — but not all — frequencies. This improvement trend was not specific

*Most of the deaths appeared to result from the inability of some of the monkeys to adapt to the caged life of the vivarium.

to any one frequency, although it was most often absent at 4,000 cps. The accompanying graphs (See Fig. 1 to 6) show these trends for each of the animals whose data were available. Joe (see Fig. 1) shows the most striking improvement

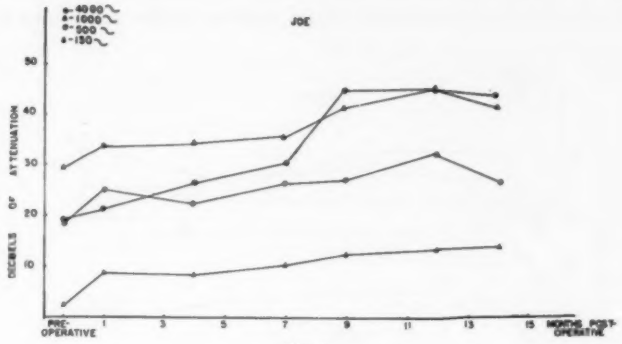

Fig. 1.

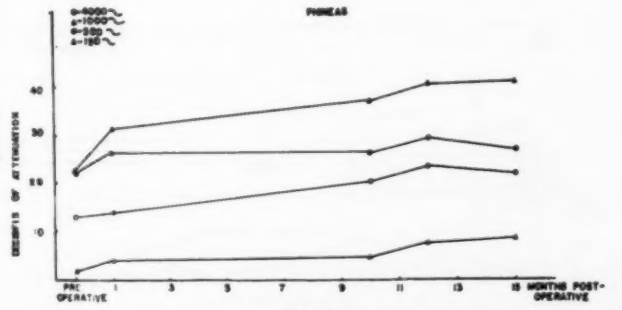

Fig. 2.

at all frequencies except 500 cps; Bennie (see Fig. 3), on the other hand, shows in general the leveling-off which had been expected; and the other animals show specific improvements at various frequencies.

For all 11 of the animals in this series the prefenestration audiogram was compared with the final postfenestration

audiogram. These final data were obtained from nine to 15 months after the fenestration except in the case of Dunois, whose final limens were obtained two years postoperatively. The differences between these two sets of limens were computed and tested for significance and are reported in Table V. Treating these data in the same way as those of Results Sec-

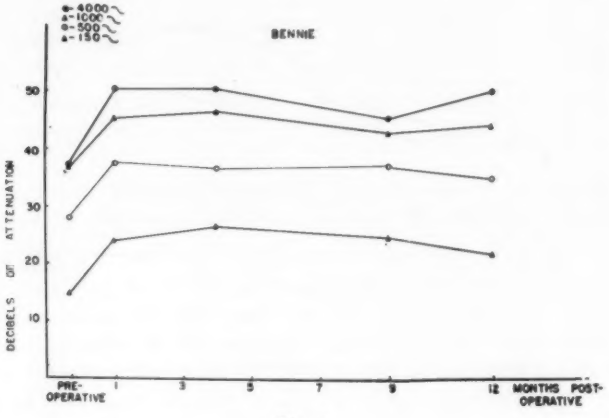

Fig. 3.

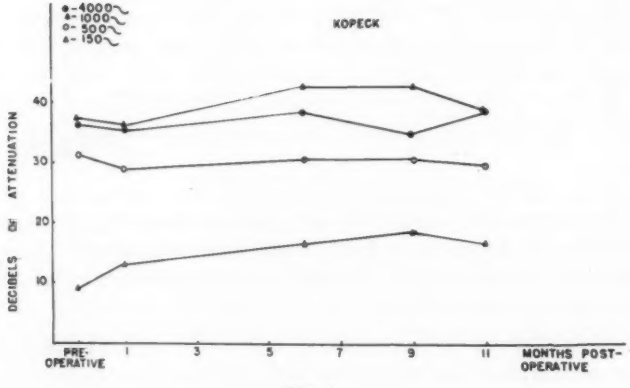

Fig. 4.

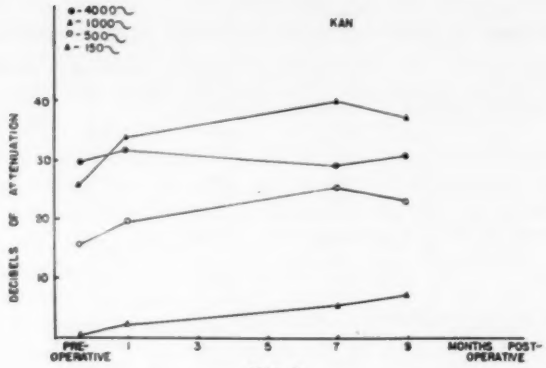

Fig. 5.

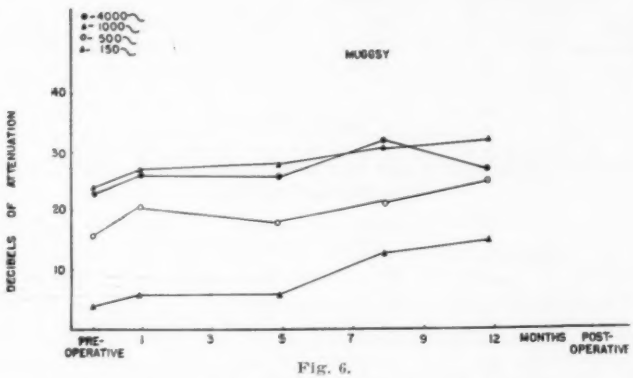

Fig. 6.

tion B, it is found that nine, or 82 per cent, of the monkeys had improved hearing for at least one frequency. Only two of the animals who showed improvement exhibited significant losses.

Table VI, again treating the data as in Section B, shows the results of dealing with each frequency separately. Here the greatest percentage of monkeys showed improvement at 150 and 4,000 cps. The greatest average improvement was at 1,000 cps.

TABLE V.

CHANGES IN HEARING MAINTAINED AT TIME OF DEATH.

Monkey	150 cps	500 cps	1,000 cps	4,000 cps
Gains at all four frequencies N-4, 36.4%				
Bennie	6.3	6.0	5.2	12.3
Joe	12.0	8.7	12.8	25.0
Muggsy	11.3	9.3	8.0	4.3
Phineas	7.0	8.6	18.0	11.3
Gains at three frequencies N-2, 18.2%				
Kan	7.3	7.5	11.4	(2.0)
Cleopatra	2.5	(4.7)	4.6	10.5
Gains at one frequency N-3, 27.3%				
Clarissa	(−1.5)	−6.0	(0.3)	6.3
Kopeck	7.5	(−2.3)	(0.5)	(2.0)
Alma	(2.2)	−6.0	4.0	(2.5)
No improvement N-2, 18.2%				
Dunois	−9.5	−5.0	−6.0	(4.7)
Angelpuss	−8.7	(1.0)	−27.7	−7.3

TABLE VI.

STATISTICS FOR THE GROUPS OF ANIMALS SHOWING IMPROVEMENT AT EACH FREQUENCY NINE TO 15 MONTHS POSTOPERATIVELY.

	150 cps	500 cps	1,000 cps	4,000 cps
Number of animals gaining	7	5	6	7
% of total N (11)	63.6%	45.4%	54.5%	63.6%
Average gain	7.7 db	8.0 db	10.0 db	9.1 db

D. Histologic Examination of the Ears: Histologic data were available for 18 of the 19 fenestrated monkeys used in Sections B and C of this experiment. The ears of Mrs. Adolph were lost, and sections of the posterior part of the fistula were missing from a few of the ears. In spite of these drawbacks, the histologic specimens for the majority of the animals were quite satisfactory.

In every one of the random sample of control ears which was examined microscopically evidences of labyrinthitis were observed in the cochlear sections. In addition to this random

sample, the control ears of all animals with marked hearing improvement which did not seem to be accounted for by the histologic condition of the experimental ear were examined. In no case was there evidence of a functional cochlea in the control ear.

When it was attempted to relate the histologic condition of the experimental ear with the monkey's functional hearing data, the temporal difficulty described in Section C was encountered. Occasionally an animal's rapidly failing health would necessitate dispatching him several months after his hearing had last been tested; however, we were able to dispatch 13 animals immediately following a postoperative limen determination. These 13 include the 11 reported in Section C and two additional animals. For the remaining animals, their most recent limens were considered in conjunction with their histologic data with due allowance having been made for the time elapsed between the last test and the animal's death.

The results of histological examination of the ears made it apparent that no easy generalizations could be made concerning the relationship between the animals' functional hearing and the condition of their inner and middle ears. No simple parallels as between open fistula and improvement in hearing and closed fistula and no improvement could be found; nor was the converse of this statement borne out by the data. Likewise, no evidences of abnormal progressive processes such as cyst-formation or adhesions to form functional connections between the disarticulated ossicles were observed in the ear sections of those animals showing progressive hearing improvement.

Table VII, which contains a brief description of the ear sections of the 13 animals dispatched immediately after a hearing test, enables one to make a comparison of the hearing function with the ear structure and shows the primary result of this study: that the structural changes in the ear (such as patency of fistula and thickness of flap) which have been assumed to be associated with improvements and losses in functional hearing do not demonstrate the high correlations that would be expected.

TABLE VII.

COMPARISON OF FINAL POSTOPERATIVE HEARING WITH STRUCTURE OF EAR

Hearing Gain at	Animal	Operative Technique			Condition of Middle and Inner Ear		
		Flap	Fistula	Packing	Flap	Fistula	Abnormalities
4 freq.	Benny	Tympano-meatal	Typical	Paraffin	Very thin	Bony closure	Drainage from ear first month postoperative
4 freq.	Joe	Plastic	Typical	Paraffin	Heavy with underlying cyst	Obliterated	Perilymphatic part of canal almost filled with bone
4 freq.	Muggsy	Tympano-meatal	Typical	Paraffin	Thin covering cystic tissue	Completely covered by thin bone	Flap had slipped loose from canal
4 freq.	Phineas	Plastic	Typical	Cotton	Good with epithelial masses near fistula	Completely closed	None
3 freq.	Kan	Plastic	Typical	Paraffin	Thin	Covered by minimal layer of bone	None
3 freq.	Cleopatra	Tympano-meatal	Typical	Paraffin	Heavy with cysts	(Not sectioned)	Inflammatory changes in region of flap
1,000 cps	Ermintrude	Tympano-meatal	Typical	Gelfoam	Fairly thin	Closed by thin bone	None
1,000 cps	Alma	Tympano-meatal	Typical	Paraffin	Heavy with cysts	Completely closed	Bony obstruction of parts of canal
4,000 cps	Clarissa	Plastic	Typical	Paraffin	Thin, well-healed	(Not sectioned)	Part of canal occluded by bone
150 cps	Kopeck	Tympano-meatal	Typical	Skin graft	Thin, excellent	Closed with thin bone	None
None	Dunois	Tympano-meatal	Cartilage stopple	Paraffin	Thin, well-formed	Partly open	Stopple in place
None	Angelpuss	Tympano-meatal	Typical	Paraffin	Very thick	Occluded	Possible labyrinthitis in cochlea
None	Agatha	Tympano-meatal	Typical	Gelfoam	Very thick	Obliterated	Epithelial masses in canal

IV — DISCUSSION.

Confronted with these incompatible results from our two methods of investigation—functional and histologic—we are forced to answer the reader's natural question as to the validity of the data themselves. Since the design of the experiment did not permit direct evidence, indirect evidence for the validity of the data is presented in the following paragraphs.

It is fairly obvious that the interpretation of the microscopic appearance of the ears is valid. In most cases the tissues were well-preserved and well-processed. And the phenomena which we sought in examination of the ear sections (*e.g.*, regenerated bone in the fistula, condition of the flap, and new connection with the disarticulated stapes to the auditory transmission system) were not ones which might be simulated by artefacts inherent in the histological processing.

Evidence that the functional hearing tests were valid is somewhat less obvious. All the tests were performed under the direction of the author, which would tend to make them more consistent. The fact that some of the animals were tested by other supervised individuals serves as a check upon the reliability of the author's own test results. The validity of our testing methods may be indirectly indicated by the similarity of our results to those of Harris.[5] When the averages of the normal monaural limens reported in Results Section A of this study are compared to Harris' average binaural limens, our limens are roughly 3 to 6 db higher (*i.e.*, poorer), which is the accepted difference between monaural and binaural hearing. In addition, the hearing losses following removal of the incus did not vary greatly among this series of monkeys and are quite similar to losses after incus removal found by Harris in another series of monkeys.[6] Thus it appears safe to assume the validity of the hearing thresholds recorded pre- and postoperatively (see Results Sections A and B).

The progressive hearing improvements reported in Section C of the Results are more difficult to accept. It might be argued that some external physical or psychological factors

inherent in the hearing test situation would account for the progressive improvement which we have noted. There are the following possibilities:

In the first place, one might consider the possibility that deterioration with time (since the tests extended over a period of five years) of the mechanical and electronic parts of the sound and timer systems had slightly changed the frequency or intensity of the tones presented or furnished a click which might have been audible to the animal and served as an additional cue; but, if this were true, all of the animals tested at a given time should have been affected equally with the most recently tested animals having the lowest or the highest limens. Since the later monkeys as a group did not have limens appreciably lower or higher than the earlier animals, the possibility of frequency or intensity changes is probably ruled out, and the possibility that the animals responded to an extraneous cue does not satisfactorily account for the variations, both in amount and frequency specificity of the progressive improvements found in each individual animal.

It is also possible that the experimenter's criteria for acceptance of the animals' responses may have become more lenient during a five-year period of constant testing. This, however, is another factor which would be assumed to apply to all of the animals at all four frequencies, which it did not.

Finally, the progressive improvements in the thresholds might be accounted for by the factors inherent in a conditioning situation of the type we have utilized.

The immediate postoperative improvements in hearing may have been occasioned by the improvement in performance with practice which occurs in any learned response even though the fenestration was always performed after the animals' preoperative limens had reached a plateau; however, this plateau probably represents the monkey's true limen since some of the monkeys never heard below this preoperative threshold even after the fenestration had been performed.

The validity of our limens may also be inferred from the comparisons previously mentioned of our limens with those of Harris, who occasionally took as many as 86 thresholds with his animals before he reached the limit which has been shown to differ not greatly from ours.

This possible practice effect might have been cumulative during the series of periodic retests, but the day-to-day fluctuations of the animals' limens were not always in a positive direction; also, the nontest intervals — two to three weeks following fenestration and two to three months between the periodic retests — would minimize this possibility. On the other hand, the phenomenon which has been described in the conditioning literature as spontaneous recovery from the inhibition of conditioning may have taken place during the period of retesting at intervals;* however, the attenuation of the conditioning prior to the interval of rest, which is a necessary condition for this phenomenon, was not observed in our animals.

If we grant that the obtained data are reliable and valid, then we must still seek an explanation for continued hearing improvement which might be either partially or wholly independent of the condition of the fenestra.

A first tentative explanation might be the behavioristic factors of adjustment of the monkey to his deafened condition and increasing attention directed by him to faint auditory stimuli. It is difficult to avoid anthropomorphism in evaluating these factors. The individual variations among the animals which would be expected with respect to this phenomenon were quite evident; but one would expect these generalized attitudes to extend equally to all four frequencies tested, and this occurred in only one animal. Postural evidences of increased attention were not sufficiently obvious to engage the experimenter's attention, and it seems too great a jump in the phylogenetic scale to look for analogies between

*See Hull, C. L.: Principles of Behavior. New York: D. Appleton-Century, 1943, p. 300.

our monkeys' hearing and the "psychological" improvement found by Silverman *et al.*[16] in their study of subjective evaluations of hearing improvement following fenestration.

A second possible explanation of our results is in terms of the changes which our operative procedures may have caused either directly or indirectly in the monkeys' auditory transmission systems. In the first place, we were not working with otosclerotic ears but with ears having an interrupted ossicular chain. Concerning the applicability of the fenestration to an ear with a mobile oval window, Rosenwasser[14] reports a theoretical discussion of the Otosclerotic Study Group, where it was stated that patients with the relatively small hearing loss of 20 to 25 db for the critical speech frequencies have usually only partial fixation of the stapes. It had been Rosenwasser's experience that patients with a small loss did not get the large improvement from the fenestration that would theoretically be expected. At that time, "Wever attempted an explanation on the basis of phase difference in the transmission of sound waves, which differ if there is a new window made in a patient with a fixed oval window and a movable round window membrane in contrast to the patient with a new window, a *partially fixed* oval window, and a movable round window membrane."[14]

Since all of our animals had entirely mobile oval windows, it was not reasonable to expect any striking improvements in their hearing following fenestration. The foregoing explanation seems, therefore, to be refuted by the data of Sections A and B, which showed a fairly large percentage of post-fenestration hearing improvements which were occasionally greater than 20 db.

A possibly more promising explanation for our results in terms of the physical characteristics of the auditory transmission system is suggested by Békésy,[1] who measured in cadavers with missing middle ear apparatus and a model of the ear the differences in sound pressure between the round and oval windows and the artificial window of fenestration. He found that these pressure differences were affected by

sound absorption and resonance in the middle ear. This middle ear absorption differed greatly in individuals because of differential pneumatization of the mastoid bone and also because of the irregular form and many niches of the middle ear cavity, whose characteristics could be greatly altered by the surgery preliminary to fenestration. He also found "in several preparations the differential sound pressure between the round window and an artificial opening at the ampulla of the horizontal canal is of the same order of magnitude as the sound pressure in the auditory canal. In this way a total hearing loss of only 30 db arises as against the normal ear. Thus, in these cases, the fenestra is a fair substitute for a functioning middle ear."[1, p. 315] If we can apply this explanation to the immediate hearing improvements found in our animals (Results Section B), we even find a rough approximation of his 30 db total loss; for our monkeys averaged close to a 10 db gain following an initial 40 db loss.

We must also attempt to account for the progressive hearing changes reported in Results Section C. Comparable data concerning progressive postfenestration hearing changes in humans have been reported by Meurman.[13] Taking audiograms at intervals over a period of either 300 or 700 days, he was able to describe five distinct courses of postfenestration hearing changes, which were similar to the changes found in our animals. He offers no explanation for these phenomena, but mentions that 20 of his patients were exceptions to the "open fistula — improved hearing" rule and its converse. Basing an explanation again on inferences from Békésy's[1] report, we can suggest that the natural processes of regenerating bone and tissue repair, proceeding after the fenestration, may have in some way *gradually* changed the physical characteristics of the middle ear cavity, altering the sound system in a manner which might vary with frequency and thus alter the functional hearing of the animals in the ways which we have reported.

These explanations might be simply tested by fenestrating monkeys and systematically varying the size of the enlargement of the middle ear cavity; and one might find some

clarification of the progressive changes with an electrical study (as in the study by Lowy[12]) if the auditory potentials could be recorded at various intervals postoperatively.

From the foregoing arguments, therefore, it can be argued that, when we accept the data of this research as valid, we are faced with results not easily explained in the light of present knowledge concerning the auditory transmission system and the results of a typical fenestration operation upon this system. The hearing improvements we found serve to indicate that the fenestration operation may serve to improve hearing by means more complex than those heretofore credited to it.

V. SUMMARY.

A. The monaural hearing thresholds of a series of monkeys were determined both before and after the ossicular chain was interrupted by removal of the incus. The hearing losses caused by this operation averaged 44.6 db with little variability for frequencies of 150, 500, 1,000 and 4,000 cycles per second.

B. A fenestration operation was performed upon the ears from which the incus had been removed and the monaural thresholds were again taken and compared with prefenestration limens. From 58 to 68 per cent of 19 animals gained an average of 7 to 9 db at each of the four frequencies tested, but the variability of the postfenestration hearing changes was very great.

C. Periodic postfenestration audiograms showed progressive changes in both positive and negative directions at some or all of the four frequencies. The variability among the animals was again very great.

D. At time of death, nine to 15 months after the fenestration operation, from 45 to 64 per cent of 11 animals had maintained hearing improvements averaging between 8 and 10 db at each of the four frequencies.

E. A microscopic examination of sections of the fenestrated

ears showed no clear-cut correspondence between ear structure at the operative site and any pattern of functional hearing changes.

F. The possibility is suggested that the enlargement of the middle ear cavity at the time of operation and the subsequent regeneration of bone and repair of tissue might account for the changes in hearing recorded.

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A DIFFERENCE LIMEN RECRUITMENT TEST AND ITS DIAGNOSTIC SIGNIFICANCE.*

JAMES F. JERGER, M.A.,
Evanston, Ill.

I –INTRODUCTION.

The discovery of the recruitment phenomenon has created an entirely new area of study in the clinical evaluation of hearing loss. In the decade following Fowler's[1] initial description, recruitment was assumed to be a concomitant feature of all "perceptive" hearing loss,[2] but in 1946 an exhaustive study by de Bruine Altes[3] revealed that the phenomenon was apparently absent in many cases of pure "nerve" impairment. The significance of this finding was illuminated in 1948 by the work of Dix, Hallpike and Hood.[4] Using the alternate binaural loudness technique, these investigators demonstrated the presence of recruitment in 30 cases of unilateral endolymphatic hydrops, but its absence, or only incomplete presence, in 20 cases of acoustic neuroma. On the basis of these findings they concluded that recruitment was present only in lesions of the sensory receptor mechanism (*i.e.*, hair cells and organ of Corti). They felt, therefore, that the determination of the recruitment phenomenon had practical diagnostic significance in distinguishing between end-organ and retrolabyrinthine lesions.

Unfortunately the clinical utilization of these important findings has been hindered by the lack of a simple, easily administered test of recruitment that is not dependent upon audiometric configuration. Although the alternate binaural loudness test is the most valid measure of recruitment, it suf-

*Summary of a Master's Thesis conducted under the direction of Raymond Carhart, Ph.D., Department of Otolaryngology and School of Speech, Northwestern University.

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fers the obvious limitation that it can be employed only where there is a substantial difference in the threshold levels of the two ears.

It has been suggested,[5,6] however, that the nature of the loudness function in an impaired ear can be indirectly inferred from the differential sensitivity to intensity change exhibited by the ear.* Specifically, it has been reasoned that an abnormally rapid acceleration in the loudness function should manifest itself in abnormally acute differential sensitivity to intensity change.

Békésy,[7] in 1947, and Lüscher,[8] in 1948, developed effective methods for the measurement of differential sensitivity. Recently, Langebeck,[9] Halm,[10] and Denes and Naunton[11] have developed variations of these methods.

It is with the Lüscher method that this study is particularly concerned. Because it requires a minimum of special equipment, and can be easily and quickly administered, the Lüscher method seems to offer the most promising possibilities as a routine clinical test. Lüscher has incorporated into the circuit of an ordinary audiometer a modulating device which produces a loudness beat by varying the intensity of the pure tone periodically.

According to Lüscher's own technique the difference limen is measured at a sensation level of 40 db. He defines the difference limen as the average value between the points where the beat is just noticeable and just no longer noticeable. Recruitment is said to be present if the difference limen is abnormally small.

By the clinical application of his simplified test, Lüscher has been able to test the hypothesis of Dix, Hallpike and Hood over a greater variety of clinical types. His results not only

*Differential sensitivity refers to the ear's ability to discriminate changes in intensity. The unit of differential sensitivity is the difference limen. It is defined as that increment in the physical intensity of a sound which a subject perceives as a change in loudness just 50 per cent of the time.

substantiate but further extend their conclusions. Lüscher has summarized his findings as follows:

> The difference limen and the recruitment phenomenon may be regarded from the topical diagnostic point of view as being equivalent . . . the difference limen appears to be dependent upon the function of the peripheral receptors, *i.e.*, the sensory cells. An impairment of the difference limen indicates damage to the organ of Corti and thus allows peripheral disorders of the neural apparatus to be recognized. According to our experiences so far, the difference limen permits us to divide disorders of the neural apparatus into three groups:
>
> *1.* Disorders of the peripheral receptor, *i.e.*, the sensory cells of the organ of Corti, which decrease in the difference limen.
>
> *2.* Retrolabyrinthine organic disorders with normal difference limen.
>
> *3.* Psychogenic hearing disorders with a high difference limen.*

In an effort to establish the validity of this test as a measure of recruitment, Neuberger[12] tested 14 unilateral loss cases by both the Lüscher and the alternate binaural loudness methods. He found a complete qualitative parallelism between the recruitment phenomenon and a decreased difference limen, and concluded that either measure was sufficiently exact for functional diagnostic purposes.

II – PURPOSE OF THE PRESENT STUDY.

The purpose of the present study was twofold: first, to determine whether a modified version of Lüscher's technique would yield results consistent with his original findings; second, to extend the exploration over a greater number of cases, including etiological types not included in his original results.

Preliminary investigation of the Lüscher technique by the author suggested that its clinical utility might be enhanced by two major revisions: *1.* changing the sensation level at which the difference limen is measured, and *2.* simplifying the procedure of test administration.

The 40 db test level has theoretical as well as practical limitations. Doerfler's[13] results show that the difference limen in the perceptively deafened ear is abnormally decreased to the greatest degree in the range from 10 to 30 db, but at the

*E. Lüscher: "The Difference Limen of Intensity Variations of Pure Tones and Its Diagnostic Significance." Jour. Laryngol. and Otol., 65:7, July, 1951, p. 510.

40 db level it is less significantly differentiated from the normal value. It seemed advisable, therefore, to test at a level in the range from 10 to 30 db; furthermore, Békésy,[7] Reger,[14] Fowler [15] and others believe that the greatest degree of recruitment occurs just at and above threshold. For these reasons it appeared that a test level as close to threshold as possible would yield the most definitive results. Accordingly a preliminary survey was conducted to determine the optimum test level. It was found that levels below 15 db were too low for accurate judgment by normal hearing subjects; therefore, 15 db was chosen as the sensation level at which to test.

A preliminary survey of Lüscher's actual testing technique suggested that the method was also clinically cumbersome and frequently indefinitive. Specifically, the introduction of modulation through the use of an interrupter switch seemed to confuse the patient and add to the variability of results. Consequently this feature of the technique was dropped. A major part of the difficulty, however, appeared to be associated with Lüscher's descending method of stimulus presentation. The progression from maximum modulation to less and finally no modulation seemed to cause the beat to perseverate in the subject's awareness. Many subjects reported the presence of the beat when no modulation was present at all. It was found that an ascending method of stimulus presentation, with progression from no modulation to full modulation facilitated the measurement of the difference limen. In this method the difference limen is defined as the increment in intensity which is just noticeable as a beat.

Thus the original Lüscher method was modified in three ways: first, the test level was changed from 40 to 15 db; second, the interruption of the stimulus was dropped; third, the ascending method of stimulus presentation was employed.

III – GENERAL TEST PROCEDURE.

Equipment: A commercially available Amplivox Recruitment Test Set, consisting of a simple amplitude modulator, calibrated in decibels of modulation, was incorporated in the circuit of an ADC 50E audiometer. The set allowed the introduction of an intensity beat, free of transients, into the out-

put of the audiometer. The degree of modulation in the beat could be varied continuously from 0 to 5 db and could be read accurately to 0.1 db.

Procedure: With the ascending method as a frame of reference, the following general test procedure was devised and applied to all cases. The subject's audiogram was first obtained. He was then instructed in the following manner:

"Now we are going to do another kind of test. You will hear the tone all the time, but it will change like this (modulation gradually introduced, then removed). Now it is a steady tone, right? (When subject replies affirmatively modulation is gradually reintroduced.) Now it is gradually changing into a beating tone. Do you hear the beating tone now? (When subject replies affirmatively modulation is removed.) All right, this is what we will do. First you will hear the steady tone. Then I will gradually change it into a beating tone. Raise your finger when you *first* hear the beat. As soon as you signal, I will take the beat out, and we will do it over again. We will try it three or four times to make sure we don't make any mistakes. Now let's try one just for practice."

The importance of careful instruction to the patient in this test cannot be overemphasized. Unless the patient understands exactly what is expected of him, the measures obtained are meaningless. Like many other psychological tests, the task must be carefully structured for the patient if meaningful responses are sought.

The subject was given one or two practice trials to familiarize him with the judgment he was to make. Then the actual test was commenced. The hearing loss dial of the audiometer was set to exceed the threshold at the frequency being tested by 15 db, then, with the tone on continuously, modulation was gradually introduced at the rate of approximately 1.9 seconds per 1 db, until the subject signalled the appearance of the beat. Three or four trials were made at each frequency. The mean value of the trials was taken as the difference limen, or "DL," for that frequency. Frequencies were tested in the order 1,000, 2,000, 4,000, 500 and 250 c.p.s., so as to maintain consistency with common audiometric procedure.

In order to clearly differentiate this method of test administration from Lüscher's more complex technique, it will be hereafter referred to as the "Northwestern DL Test."*

*Thus, "DL" subsequently refers to the difference limen for intensity change as defined by the method outlined above, and the phrase "Northwestern DL Test" refers to the general test procedure.

IV – STANDARDIZATION.

In order to establish normative data with which to evaluate results obtained on hearing loss cases, DLs were obtained on 39 normal ears. Each ear was tested at the five frequencies 250, 500, 1,000, 2,000, 4,000 c.p.s., at the 15 db sensation level. The average DL and its standard deviation for each frequency is shown in Table 1.

TABLE 1.

AVERAGE DL AND STANDARD DEVIATION YIELDED BY 39 NORMAL EARS AT EACH FREQUENCY FOR THE 15 DB SENSATION LEVEL.

Frequency	250	500	1,000	2,000	4,000
Average DL	2.0	1.8	1.7	1.7	1.6
Standard Deviation	.28	.41	.41	.35	.40

In order to facilitate the interpretation of results obtained on hearing loss cases it seemed desirable to translate the normative data into a form or chart on which clinical results could be recorded graphically for rapid evaluation. Accordingly a chart was prepared. The chart is reproduced in Fig. 1. The test frequency is represented on the horizontal dimension and DL size, in db, along the vertical scale. The average DLs for normals at the various frequencies are shown by the heavy solid line running across the center of the chart. The broken lines on either side are separated by two standard deviations from the line showing the average normal values. The two broken lines thus enclose an area of four standard deviations. This range of four standard deviations was arbitrarily taken as the area in which normal DLs were to be expected. The choice of this range of four standard deviations was strengthened by the fact that all the original DLs from the normal hearing group fell within the area. The chart is called the "Intensity DL Record." It is used to plot the DLs yielded by a clinical case. The procedure is similar to the one employed in drawing an audiometric curve. The DL at a particular frequency is recorded on the chart as an X for the left ear and a 0 for the right ear. These points, thus drawn, are then joined by line segments. For purposes of

illustration, Fig. 1 includes a typical clinical record. The results were obtained on an ear with marked recruitment which was diagnosed as having hearing loss due to endolymphatic hydrops.

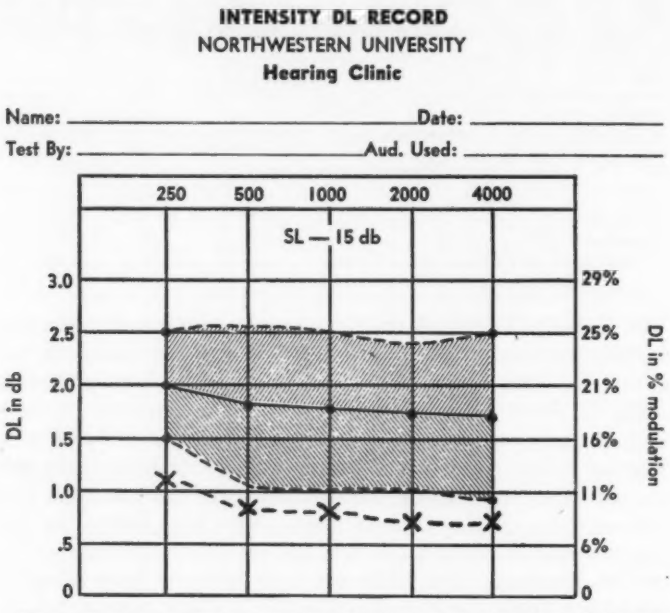

Fig. 1. Chart for recording results of DL Test when administered at a sensation level of 15 db Average value for normal DLs (●———●); limits of normal DLs (- - - -); a clinical case of endolymphatic hydrops illustrating abnormally small DLs (X - - - X).

V – EXPERIMENTAL FINDINGS.

In order to determine whether the Northwestern DL Test would yield results consistent with Lüscher's original findings, the test was administered to 89 hearing loss patients over a period of approximately one year.

Each case was diagnosed by an otologist either prior or subsequent to administration of the test. In no case did the

DL Test results influence the otological diagnosis. Diagnoses have been divided into three categories: definite, probable, and suspected, according to their degree of certainty.*

The 89 hearing loss cases were divided into two groups on the basis of the otological diagnosis. One group was composed of those cases in which the otological diagnosis indicated a pure conductive loss. The second group consisted of the remainder of the cases, *i.e.*, those in which the otological diagnosis indicated a sensori-neural component.

In analyzing the experimental data, DLs were interpreted as normal if they fell within the "normal" area of the DL Record, but abnormally small or abnormally large if they fell below or above that area respectively. This analysis applied to the majority of cases tested. As the experiment progressed, however, it became apparent that a small number of subjects, particularly those of advanced age, yielded results inconsistent with the above analysis. Their DL curves showed the expected configurations, but the curves as a whole seemed to be displaced upward, suggesting slightly larger normal DLs than the average value. For example, a case with normal hearing at 1,000 c.p.s. and a 50 db loss at 2,000 c.p.s. showed DLs of 2.4 at 1,000 and 1.1 at 2,000 c.p.s. Although the DL of 1.1 failed to fall below the "normal" area of the DL Record, it was clearly abnormally reduced relative to the DL at 1,000 c.p.s. In such cases it was decided that a DL would be interpreted as abnormally small if it deviated from the trend of the DLs which the subject obtained at other frequencies by an amount exceeding the two standard deviation distance for normals at its frequency. Similarly, in cases of unilateral loss, DLs on the impaired ear were interpreted as abnormally small if they fell below the DLs for the normal ear by the two standard deviation distance, regardless of the position of the DLs on the DL Record. As stated above, this interpretation applied to only a few cases.

*The majority of diagnoses were made by Dr. G. E. Shambaugh, Jr., Department of Otolaryngology, Northwestern Medical School, to whom the writer is further indebted for helpful suggestions throughout the study.

RESULTS.

Fifteen cases comprised the pure conductive loss group. The etiological breakdown was as follows: seven otosclerosis, five otitis media, one perforated tympanic membrane, one probably congenital stapes fixation, one unknown. In no case did the DL at any frequency fall below the "normal" area of the DL Record. The average DLs at each frequency are shown in Table 2, where the values for normal hearers are repeated for comparative purposes. The parallelism of these results demonstrates that at equivalent sensation levels the DL was normal in pure conductive hearing loss. This finding indirectly supports the hypothesis that the recruitment phenomenon and DL size are related. Since a pure conductive loss merely attenuates the energy of the sound wave as it passes through the middle ear into the labyrinth, the loudness recruitment phenomenon should not be present in such cases. This is exactly the situation which was found to exist in the pure conductive loss group.

The remaining 74 cases comprised the sensori-neural loss group. Included in this group were all cases showing a sensori-neural component whether there was a conductive component or not. The DLs exhibited by these cases, at the frequencies at which there was a sensori-neural component, could be divided into three categories:

a. Abnormally small DLs.

b. Normal DLs.

c. Abnormally large DLs.

Table 2 shows the average DL at each frequency for each one of these categories, as well as the average DLs for the pure conductive loss group and for normal hearers.

Tables 3, 4 and 5 show the etiological classifications, and the number of cases in each classification, falling under each of the three categories of DL size exhibited by the sensori-neural loss group. The number of cases in each etiological classification is further subdivided into three groups according to the certainty of diagnosis.

TABLE 2.
AVERAGE DL AT EACH FREQUENCY FOR NORMAL HEARERS, PURE CONDUCTIVE LOSS GROUP, AND SENSORI-NEURAL LOSS GROUP.

Freq.	Norm. Hear.	Cond. Loss	Sensori-Neural Loss		
			Abnormally Small DL	Normal DL	Abnormally Large DL
250	2.0	2.0	1.0	2.0	4.2
500	1.8	2.0	.9	1.8	4.2
1,000	1.7	1.9	.9	1.8	3.6
2,000	1.7	1.9	.9	1.8	4.3
4,000	1.6	1.8	.9	1.6	3.3

TABLE 3.
NUMBER OF CASES IN EACH ETIOLOGY SHOWING SMALL DIFFERENCE LIMENS, CLASSIFIED BY CERTAINTY OF DIAGNOSIS.

Etiology	Definite	Probable	Suspected	Total
Acoustic Trauma	3	5	—	8
Endolymphatic Hydrops	6	2	1	9
Hereditary	1	4	—	5
Skull Trauma	—	3	—	3
Otosclerosis with secondary nerve degeneration	13	—	—	13
Meningitis	2	—	—	2
Otitis Media with secondary nerve degeneration	4	—	—	4
Scarlet Fever	2	—	—	2
Influenza	—	1	—	1
Measles	—	1	—	1
Mumps	—	1	—	1
Maternal Rubella in Pregnancy	—	1	—	1
Thrombosis of internal auditory artery	1	—	—	1
Presbyacusis	1	1	—	2
Total				53

Each of the three categories of DL size will now be discussed separately.

1. Sensori-Neural Losses with Abnormally Small DLs:

Table 3 lists the etiological classifications in which the DLs were found to be abnormally small for the frequencies at which there was a sensori-neural component. The group con-

TABLE 4.
NUMBER OF CASES IN EACH ETIOLOGY SHOWING NORMAL DIFFERENCE LIMENS, CLASSIFIED BY CERTAINTY OF DIAGNOSIS

Etiology	Definite	Probable	Suspected	Total
Presbyacusis	4	4	—	8
Streptomycin	1	1	—	2
Cerebellopontine angle lesion	1	1	—	2
Psychogenic	1	—	—	1
Psychogenic Overlay	—	—	1	1
Total				14

TABLE 5.
NUMBER OF CASES IN EACH ETIOLOGY SHOWING ABNORMALLY LARGE DIFFERENCE LIMENS, CLASSIFIED BY CERTAINTY OF DIAGNOSIS.

Etiology	Definite	Probable	Suspected	Total
Psychogenic Overlay	—	—	4	4
Malingering	—	—	1	1
Brain Damage	1	—	1	2
Total				7

sists of eight acoustic trauma losses, nine cases of endolymphatic hydrops, five cases of presumably hereditary loss, three cases of probable skull trauma, 13 cases of otosclerosis with secondary nerve degeneration, two losses from meningitis, four cases of otitis media with secondary nerve degeneration, two scarlet fever losses, two cases of presbyacusis, one case of nerve deafness from influenza, measles, mumps, and maternal rubella in pregnancy, and one vascular lesion of the cochlea.

These findings are in agreement with Lüscher's report that the DL is abnormally small in acoustic trauma, Ménière's disease and endogenous deafness. In addition, they include other etiological types not included in Lüscher's original results.

The findings reported in Table 3 have important clinical and theoretical implications. For example, the apparent presence of reduced DLs in nerve loss secondary to otosclerosis

suggests a method for estimating the cochlear reserve of the otosclerotic. Information on this question is an important consideration in prognosis for the fenestration operation. The level of the bone threshold in otosclerosis is not an adequate index of cochlear function because of the mechanical shift in bone acuity occasioned by the immobilization of the oval window (the "Carhart Notch"); but if the 13 otosclerotic cases reported in Table 3 are typical, the presence of an abnormally small DL apparently indicates cochlear invasion which could have been estimated without recourse to the bone conduction measurements.

From the theoretical standpoint, detailed knowledge of the nature of the pathological lesion in many clinical conditions has been hampered by the inability to perform detailed histological investigation of human subjects at autopsy. For example, in a recent review of the literature on hereditary deafness, Kinney[16] found only one report of a human subject with histological correlates in this country. The vast bulk of our knowledge of hearing loss due to heredity, toxic invasion of the cochlea, etc., has, of necessity, come from animal experimentation; but by testing for the recruitment phenomenon it may be possible to infer the locus of pathology in the living subject, thus adding significantly to the understanding of auditory impairment.

2. Sensori-Neural Losses with Normal DLs:

Table 4 lists the etiological classifications in which the DLs were found to be normal. The group consists of eight cases of presbyacusis, two cases of streptomycin damage, two cases of cerebellopontine angle lesion, one case of psychogenic loss, and one case of suspected psychogenic overlay.

These findings are in agreement with Lüscher's report of normal DLs in cerebellopontine angle tumor, streptomycin damage, and disorders of the central nervous system.

The number of presbyacusics observed to have normal DLs is sufficiently large to warrant special comment. Eight of the 10 presbyacusics included in the present study showed normal

DLs, while the other two had abnormally small DLs. This finding might be explained in several ways. There is always the possibility that the two cases with small DLs represent erroneous diagnoses, but there is also the possibility that there may be more than one kind of presbyacusis. A third possibility is that the primary site of pathology may be in the spiral ganglion with variation in the degree of cochlear damage, depending upon the severity of the lesion.

At any rate it would appear that recruitment is the exception rather than the rule in presbyacusis if one may judge from the 10 cases we have observed. This finding has important implications for the fitting of hearing aids on these cases. It has been commonly observed that presbyacusics often present serious tolerance problems for loud sounds in hearing aid use. This has been inferentially attributed to the recruitment phenomenon. Limitation of maximum power output, and, more recently, compression amplification, have, therefore, been used in an effort to counter the tolerance problem; but the apparent absence of the recruitment phenomenon in most presbyacusics suggests that the nature of the tolerance problem is not yet fully understood. If it is a central rather than peripheral phenomenon, our present methods of handling the problem may be entirely inadequate.

The apparent absence of recruitment in the cerebellopontine angle lesion cases has obvious implications in the differential diagnosis of this condition, especially in the early stages.

3. Sensori-Neural Losses with Abnormally Large DLs:

Table 5 shows that in seven cases the DLs were abnormally large. Four of these cases were suspected psychogenic overlay, one was a suspected malingerer, and two were cases of central nervous system damage. Because of the small sample size and the absence of neuropsychiatric evaluation of these patients, specific conclusions are not warranted. It is significant, however, that in the application of the DL Test to a wide variety of cases a limited number showed atypical re-

sponses in the form of abnormally large difference limens. Whenever this occurred there was sufficient concomitant evidence to lead to the tentative conclusion that the patient's apparent hearing loss was not on a peripheral organic basis.

Generalizations based on this finding must await further controlled exploration. At the present writing it can only be stated that abnormally large differential sensitivity to intensity change apparently does not fit the total clinical picture of peripheral organic involvement of the auditory mechanism.

Summary of Clinical Findings:

It is apparent from the data reported above that the relationship between sensori-neural hearing loss and the recruitment phenomenon is more complicated than has previously been supposed. Our findings show that in pure conductive hearing loss the DL is normal, indicating the absence of recruitment, but in sensori-neural loss the DL may be abnormally small, indicating recruitment, or normal, indicating the absence of recruitment. A small number of cases tested showed abnormally large difference limens. In each one of these cases there was sufficient concomitant evidence to lead to the tentative conclusion that the patient's apparent hearing loss was not on a peripheral organic basis. No specific conclusions regarding this apparent relationship are herein drawn.

Validation of the DL Test as a Measure of Recruitment:

In order to demonstrate that the DL Test is a valid test of recruitment, the alternate binaural loudness test was administered in addition to the DL Test wherever possible. It was reasoned that if reduced DLs were a concomitant feature of the recruitment phenomenon, then the two tests should yield equivalent results. It was possible to apply both tests to nine of the cases in the sensori-neural loss group. In all cases the two explorations yielded equivalent results. This finding was interpreted as further substantiation of the parallelism of abnormally small DLs and the recruitment phenomenon.

Clinical Utility of the DL Test:

During the period of approximately one year that the DL Test has been routinely administered in the two Northwestern University Hearing Clinics, its clinical significance for otologic practice has become evident in a number of areas.

Practical applications of the test as an index of cochlear function in the otosclerotic, and as an aid in the differential diagnosis of retrolabyrinthine losses have been described above.

In general, the value of the DL Test lies in its capacity to facilitate diagnosis by adding confirmatory evidence in difficult cases. For example, the otologist is often confronted with unilateral perceptive loss cases in which the history and standard test findings fail to differentiate between etiologies yielding similar audiometric configurations, as, for example, acoustic neuroma and endolymphatic hydrops. In such cases the DL Test effectively differentiates the two etiologies. It has been our experience that hydrops cases show sharp, consistent responses to abnormally small changes in intensity of the order of 0.8 to 0.9 db, but acoustic neuroma cases do not respond to intensity changes until they reach the normal value of 1.6 to 2.0 db. There is both a quantitative and qualitative difference in the test results. Not only are the DLs in the hydrops cases smaller than normal, but the subject's responses are sharper, more consistent, less variable than the DLs in the neuroma cases.

VI – SUMMARY AND CONCLUSIONS.

The Northwestern DL Test, a simplified revision of the Lüscher difference limen test for recruitment, was first standardized on 39 normal ears, then administered to 89 hearing loss cases of varied etiology. Pure conductive losses showed normal DLs, but in sensori-neural losses DLs were abnormally reduced in some cases, normal in others, and abnormally large in still others. Examination of the etiological classifications falling under each of these categories suggests the following conclusions:

1. The Northwestern DL Test yields results consistent with Lüscher's more complex technique. This fact leads to the conclusion that the new technique is clinically preferable, since it allows the test to be administered to a greater range of hearing loss cases. (The Lüscher technique cannot be used if the loss by air conduction is greater than 60 db.)

2. DLs were found to be abnormally small in those etiological classifications in which histopathological evidence suggests damage to the sensory receptor mechanism, *e.g.*, acoustic trauma and endolymphatic hydrops. This fact leads to the conclusion that DLs are abnormally small in end-organ damage.

3. DLs were found to be normal in hearing loss due to lesions of the cerebellopontine angle. This fact leads to the conclusion that differential sensitivity to intensity change is normal in retrolabyrinthine lesions.

4. Abnormally poor differential sensitivity to intensity change apparently does not fit the clinical picture of simple peripheral involvement of the auditory mechanism. This finding emphasizes the fact that in the administration of the DL Test the clinician must be alert to the possibility of atypical results.

5. DL Test results and alternate binaural loudness test results were equivalent in all cases tested. We may conclude, therefore, that abnormally acute differential sensitivity to intensity change is an inherent feature of the loudness recruitment phenomenon.

6. Application of the DL Test to a wide variety of clinical cases suggests that the test, as a measure of recruitment, has important implications, both as a clinical and laboratory tool.

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A CLINICAL AND RESEARCH VERSION OF THE BEKESY AUDIOMETER.*

SCOTT N. REGER, Ph.D.,
Iowa City, Iowa.

In 1947, Bekesy[1] described a new type of a continuously variable-frequency, self-testing, automatic-recording audiometer which obviously possesses several features of outstanding merit. This audiometer consists essentially of *1.* a continuously variable audiofrequency generator, the frequency of which is varied over the desired part of the frequency spectrum by means of a motor drive; *2.* an instantly reversible motor-driven attenuator by means of which the intensity of the test tones can be increased or decreased as desired with a contact key; and *3.* a recording pen which indicates the intensity level of the attenuator on the Y axis (ordinate) of an audiogram blank which is moved along its X axis (abscissa) in synchronization with the change in frequency of the continuously variable audiofrequency generator.

The listener automatically records his threshold acuity by alternately pressing a key until the tone is just perceived and releasing the key until the tone is just inaudible, while the frequency is varied slowly over the test frequency range by the motor drive. (The key can be connected so that the listener can be instructed to press the key only as long as the test tones can be heard.) This procedure not only measures the threshold intensity value but also gives an indication of the intensity difference limen at or near the threshold level, which inherently demonstrates the presence or absence of recruitment at threshold in the pathological ear. Bekesy points out that since in this audiometer the test tone is always in the vicinity of the threshold, little time is lost in the presentation of subliminal stimuli.

*Department of Otolaryngology and Oral Surgery, University Hospitals, State University of Iowa, Iowa City, Iowa.

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In addition to these features, the Bekesy audiometer appealed to the writer because of an interest in "objective" and standardized audiometry.[2] With this audiometer the testing skill of the examiner is limited to instructing the patient how to take the test and to adjusting the instrument's controls — operations which can be performed satisfactorily by any novice in audiometry. The audiometer also appears to be a superior research and teaching tool for the study of certain hearing problems, some of which will be mentioned later. These considerations proved to be sufficiently motivating to induce the writer to attempt the construction of a Bekesy Audiometer.* The resulting instrument has proved exceedingly satisfactory, and the early experimental and clinical results sufficiently encouraging to warrant a report on the construction details and research and clinical possibilities of the instrument at the present time.

PERFORMANCE CHARACTERISTICS.

The new instrument bears very little physical resemblance to the photograph of the original Bekesy Audiometer (not shown in the present paper), which was a laboratory model devised by Bekesy to test the practicability of his new concept for testing auditory acuity.

Fig. 1 shows the appearance of the instrument constructed by the writer. The continuously variable audiofrequency generator on top of the cabinet is a General Radio Co. (Cambridge, Mass.) type No. 1304-A Beat Frequency Oscillator. The frequency change of this particular oscillator is a logarithmic function per degree rotation of the frequency control dial above 100 cycles per second. The output level meter under the left end of the oscillator provides an instantaneous check on the output intensity level of the oscillator whenever desired. The audiogram blank is clipped on a flat audiogram carriage board which is driven from right to left in synchronization with the rotation of the frequency control dial of the oscillator. The instrument illustrated can be used to

*The author was ably assisted in the construction of the instrument by Mr. Floyd L. Rinehart, electronic technician in the Department of Otolaryngology.

test the acuity over any five consecutive octaves between 100 and 20,000 cycles per second. The audiogram carriage board is geared to move a distance of 28 mm. per octave at any one of three different testing time intervals — one minute, two minutes or four minutes per octave. This enables the acuity to be tested over the significant five-octave frequency range of 250 to 8,000 cycles per second in five, 10 or 20 minutes as desired. A special gear shift control permits a secondary

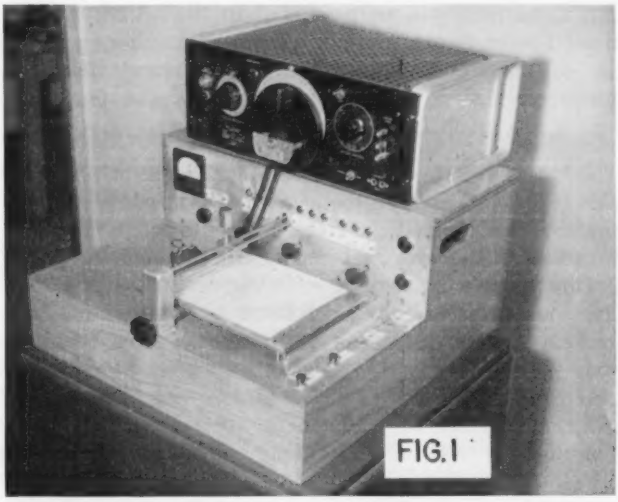

FIG. 1

reduction in the rate of the frequency change of the oscillator by a factor of five, thereby permitting any desired octave interval to be spread out over the space and time ordinarily occupied by five octaves. This permits a detailed study of sudden and marked acuity changes that occur within relatively narrow frequency ranges or intervals.

The instantly reversible motor drive to the attenuator is geared to attenuate the signal at the rate of 2, 1 or 0.5 db per second as desired. A maximum attenuation of 2,400 db per octave can be obtained by using a combination of the octave spread feature, the longest testing time (20 minutes) and the

maximum signal attenuation (2 db per second). Almost any desired combination of frequency range, testing time, attenuation in decibels per second, and size and shape of audiogram blank can be obtained by selection of appropriate gear ratios and other mechanical parts when the audiometer is constructed.

Since it is assumed that the clinician will be little interested in the mechanical construction details of the instrument, these data will be presented in the latter part of the article; however, one mechanical detail simplifies and facilitates so many of the features of the new Bekesy-Type Audiometer that a general description of the part and its function are given immediately below.

The "thing" in question is a one-foot length of pinion wire, which is an elongated 16-tooth gear, two-thirds of an inch in diameter. The knob on the front and center of the audiometer is attached to the proximal end of the pinion wire (see Fig. 1). This pinion wire may be motor-driven at any one of three revolutions rates or may be rotated by hand if desired. A spur gear on the distal end of the pinion wire (behind the control panel) engages the mechanism which drives the oscillator frequency control dial. The pinion wire also propels the audiogram carriage board by contact between the teeth of the pinion and the teeth of racks along the front and back edges of the board. The rotation of the pinion wire, therefore, mechanically synchronizes the rate of frequency change of the oscillator and the speed with which the audiogram carriage board is moved. Calibration of the audiometer so that the theoretically "normal" curve appears as a straight line (at the present time assumed to be at or near the mean distance or range between the extreme excursions of the recording pen) also is accomplished by the driving force of the pinion wire, as described later in the construction details.

A gear shift mechanism is mounted on the front panel (just to the right of the pinion wire), by means of which the mechanism which drives the pinion wire can be disengaged or the speed of rotation of the pinion be varied by the motor

drive. Disengaging the motor drive to the pinion permits it to be rotated by hand to facilitate resetting the synchronized positions of both the oscillator and the audiogram carriage board to their respective starting points for additional tests. The entire acuity test can be given with manual rotation of the pinion wire to decrease the testing time whenever desired. Since rapid changes in acuity occur less frequently at the lower frequency levels, the time required to give the entire test can be shortened by rotating the pinion manually as rapidly as desired, up to a given frequency level, at which point the pinion can be rotated slower or the motor drive can be engaged. Manual rotation of the pinion also permits rapid measures of acuity at octave and semioctave levels if desired.

A reasonably effective flexibility of control is incorporated in the present instrument. The attenuation rate (in decibels per second) can be varied at any time during the test procedure. The slowest rate (0.5 db per second) should be used while measuring recruitment and while testing individuals who have slow reaction times. The testing time (or rate of frequency change) can be varied at any instant so that the lower frequencies can be tested rather rapidly (one minute per octave) and the higher frequencies, where sudden changes in acuity are most likely to occur, can be tested more slowly (four minutes per octave). The driving mechanism between the pinion wire and the frequency-changing dial of the oscillator can be disengaged and set by hand so that the acuity can be tested at any frequency level for as long as 20 minutes if desired. As previously indicated, the octave-spread reduction gear permits the acuity for any single octave to be tested over any one of three different time intervals — five, 10 or 20 minutes — and at any of three different attenuation rates — 2, 1 or 0.5 db per second.

CLINICAL APPLICATION.

Many of the clinical uses of this unique type of audiometer were mentioned in Bekesy's[1] paper. Any other uses that may be suggested by the present writer probably appeared so obvious to Bekesy that he did not consider them worthy of com-

ment; nevertheless, a few of the more common uses and advantages of the instrument will be indicated immediately below, since the original articles in the *Acta Oto-Laryngologica* may not be readily available to those who may be interested in this type of audiometer.

As is apparent, the Bekesy Audiometer threshold acuity test more closely approximates a "standardized" hearing test than does conventional manual audiometry, since the audiometrist has nothing to do with the presentation of the test stimuli after the instructions have been given to the patient and the instrument turned on to start the test. Since the patient's reactions to the test are visible to the audiometrist and also recorded on the audiogram blank, the patient's ability to follow the test instructions (take the test) is soon revealed.

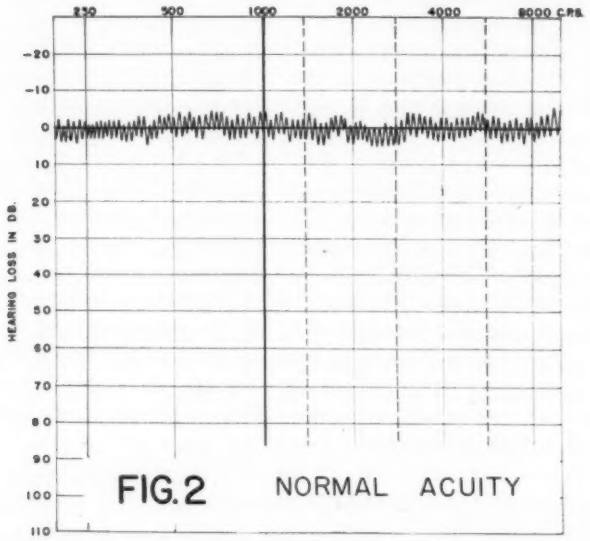

Fig. 2 shows an audiogram of a "normal" ear of a 20-year-old university student. The intensity range at threshold — the intensity difference between the maximum and minimum

levels about the threshold value as indicated by the peak and valley recordings of the up and down excursions of the recording pen — approximates a value of 6 to 8 db. This 6 to 8 db "normal" range is also approximated by individuals with middle ear pathologies and by those with "neural" hearing impairments in which recruitment is absent. Individuals who exhibit an intensity range twice as great as that of the average normal ear are not capable of taking the test with a desirable degree of accuracy; nevertheless, it is possible that the mean value of the midpoints between the peaks and valleys of wide pen excursions does indicate a usefully accurate threshold value. Many children between eight and nine years of age have been tested who exhibit intensity ranges about threshold of less than 10 db.

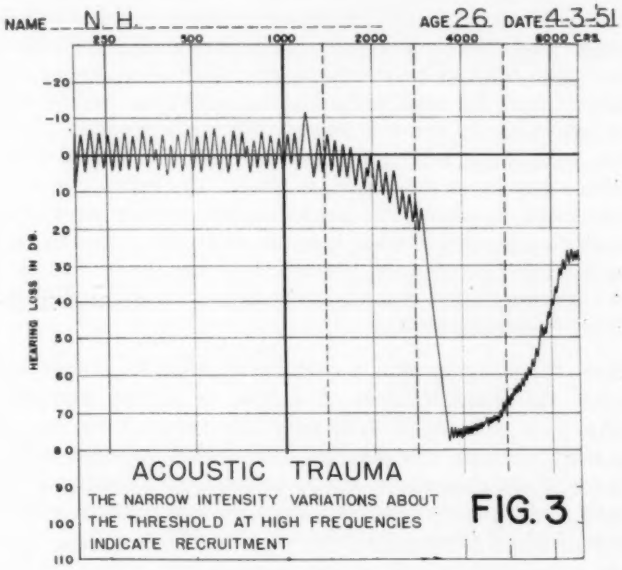

Fig. 3 is an audiogram of one ear of an individual with normal acuity for low and a loss for high frequencies. The intensity range about the threshold value is "average" over

the frequency levels at which the acuity is normal but is markedly reduced at the frequency levels at which the maximum hearing loss exists. This reduced intensity range, of course, is due to the fact that the patient experiences a disproportionately rapid increase in loudness as the intensity is raised above the threshold value. As pointed out by Bekesy, this method of measuring threshold acuity automatically and inherently demonstrates the presence of recruitment, since the intensity difference limen near the threshold is more acute in individuals who have recruitment. This test provides one of the easiest, quickest and most accurate techniques for detecting recruitment. No additional time, equipment or confusing instructions or tasks are involved. Any child or adult capable of taking the test unknowingly tests himself for recruitment over the test frequency range. The test can be applied to all types of hearing impairments, including unilateral, bilaterally symmetrical high frequency losses and bilaterally symmetrical flat losses. Appropriate masking must be used while testing unilateral impairments. It is advisable to use the slowest attenuation speed (0.5 db per second) while testing individuals with recruitment as well as those who have slow reaction times. In certain cases with recruitment in which the acuity varies rapidly as the frequency changes it is advisable to ascertain the intensity range at a few constant frequency levels, for example, at 500, 2,000 and 4,000 cycles per second, using the same attenuation rate at each frequency level.

Since the instrument is a continuous frequency range audiometer, the characteristics of sudden or abrupt changes in acuity with changes in frequency are revealed by the test. The most accurate and detailed measure of extremely abrupt or precipitous changes in acuity is obtained by using a combination of the octave-spread gear reduction drive and the slowest testing time — 20 minutes per octave.

The presence of occasional high peaks or spikes in the intensity range usually indicates the frequency or frequencies at which tinnitus is confused with the test tones, thereby indicating the frequency of the tinnitus. The instrument can

be equipped and calibrated for use with a bone conduction vibrator for obtaining bone conduction acuity measurements if desired.

RESEARCH APPLICATIONS.

The Bekesy Audiometer is an excellent device for use in any research problem in which the measurement of auditory acuity for pure tones is involved. The instrument is peculiarly suited for use in studies in which it is desirable to measure acuity over a continuous frequency range instead of octave or semioctave frequency levels and in problems in which the test tones are presented for relatively long periods of time. This audiometer is ideal for measuring acuity under test conditions which alter the threshold sensitivity with time. For example, it is invaluable for studying the factors of attention and "fatigue" over relatively long intervals, and for studying in detail the rate of recovery of acuity after the ear has been exposed to exceedingly high noise levels; however, several testing features of the "instrument-patient" combination need early investigation. For example, does the correct threshold lie at the midpoint between the extreme intensity range which is recorded by the peaks and valleys of the excursions of the recording pen? What is the effect of practice? What are the effects of different testing times (rates of frequency change) and of different attenuation rates on the range of the intensity variation above and below the correct threshold value?

CONSTRUCTION HINTS.

Sufficient descriptive and photographic data are given below to enable a competent mechanical-electrical engineer to construct a Bekesy Audiometer. The instrument presents more mechanical than electrical problems. As indicated previously, almost any desired combination of frequency range, testing time, attenuation rate in decibels per second, and size and shape of audiogram blank can be obtained by selection of appropriate gears and other components at the time the audiometer is constructed.

The general appearance of the instrument is shown in Fig. 1. The chassis is constructed of sheet aluminum and plywood. The plywood base is 23 inches wide and 23½ inches deep. The front of the base is 5 inches high and the back of the base, which supports the oscillator, is 12½ inches high. The control panel, on which the meter and gear shift mechanisms are mounted, is 7½ inches high. The audiogram carriage board is 12 inches long and 8½ inches wide.

Fig. 4 shows some of the detail of the pinion wire gear (24 pitch, 16 teeth), the audiogram carriage board and the recording pen carriage mechanism. Twenty-four pitch racks

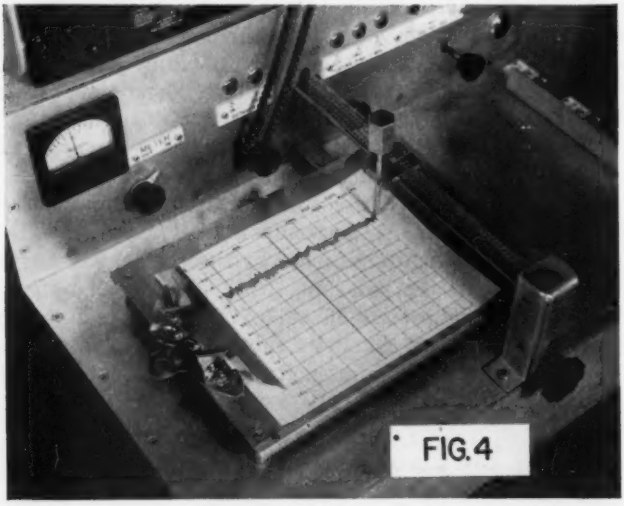

FIG. 4

extend along the entire length of the front and back edges of the audiogram carriage board. The pen carriage is moved by an endless ladder chain (No. 1A) driven by a two-inch pitch diameter sprocket which is attached to the attenuator shaft. The diameter of this sprocket and the number of degrees of rotation of the attenuator shaft which produce the desired total attenuation range determine the length of the Y-axis or height of the audiogram blank. The proximal end

of the ladder chain is supported by a seven-teeth idler sprocket. The attenuator in the model illustrated covers a total attenuation range of 140 db in 2 db steps. Additional models are being constructed with stepless or continuously variable intensity attenuators. Red and blue ink ball point pens have been found reasonably saatisfactory for use as the recording pens. The hexagonal shaped piece of brass on top of the pen has a hole in the bottom slightly larger than the diameter of the pen filler which fits over the top of the pen to provide sufficient pressure on the point to cause it to record.

Two separate but almost identical gear shift mechanisms are used to drive the attenuator and the synchronized oscillator-board carriage mechanism. Fig. 5 shows a block dia-

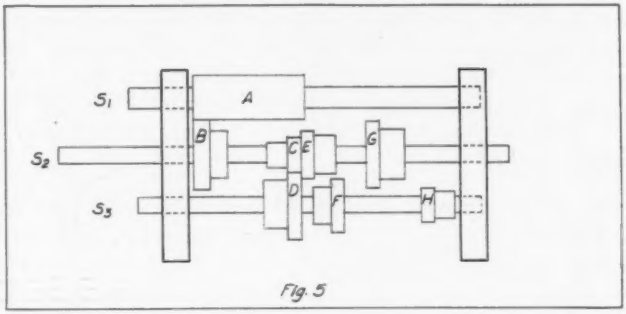

Fig. 5

gram of the essential features of these mechanisms. Shaft S1 is the driver shaft and consists principally of a two-inch length of pinion wire (24 pitch, 16 teeth), gear A. S2 is the gear-shift idler shaft, which is shifted back and forth to obtain the three different speeds of rotation of the driven shaft, S3. Gear B is a 24 pitch, 24 teeth spur gear, which is always in mesh with A. All the remaining gears of Fig. 5 are 32 pitch spur gears. Gears C and H have 16 teeth, E and F have 24 teeth and G and D have 32 teeth.

When S2 is in the position shown, with gears C and D meshed, S3 is driven with the slowest speed, X. When S2 is positioned so that E and F mesh the speed of rotation of S3 is

2X and when G and H are meshed the speed rotation of S3 is 4X. The gears on S2 and S3 are so spaced in relation to each other that S2 has two different neutral positions — positions at which none of the six speed-ratio gears is meshed. When the gear shift idler shaft associated with the attenuator is in a neutral position, the attenuator can be rotated manually if desired by means of an extension from the attenuator shaft to the outside of the cabinet. When the gear shift idler shaft associated with the oscillator-audiogram carriage board mechanism is in neutral, the position of the audiogram carriage board can be varied or reset manually by means of the knob of the proximal end of the pinion wire.

The shafts of the gear assemblies are at right angles to the control panel of the cabinet shown in Fig. 1. The two knobs just above the back edge of the audiogram carriage board (see Fig. 1) are attached to the gear shift idler shafts. These shafts are held in their meshed and neutral positions by means of spring detents which make friction contact with appropriately spaced grooves cut in the circumference of the shafts.

Fig. 6 is a photographic reproduction of the gear shift mechanisms of a later model than that shown in Fig. 1. In the later model the shafts are parallel with the front of the control panel, the lower part of which can be seen on the right side of the photograph. P1 and P2 are 12 teeth sprockets, driven by $\frac{1}{4}$-inch No. 25 chains to sprockets on the shafts of the motors mounted under the aluminum plate to which the gear mechanisms are attached. Gear I, attached to the attenuator shaft, is a 32 pitch, 96 teeth spur gear, driven by a 16 teeth spur gear (H of Fig. 5) on shaft S3 of the gear shift mechanism on the left of Fig. 6. Only the hub of the sprocket (on the attenuator shaft also) which drives the ladder chain to the pen carriage is visible. Only the lower loop of the chain can be seen.

In Fig. 6 the gear-shift idler shafts are positioned by means of rods R1 and R2, which project through horizontal rectangular openings in the control panel. Tension springs

T1 and T2 hold the rods in their desired positions in appropriately spaced slots in the lower borders of the rectangular openings. F1 is the fulcrum of rod R1 and F2 is the fulcrum of R2. M is a rectangular piece of brass, rigidly attached at the top to the end of the lever actuated by R2. Shaft S2 extends through a hole in the lower end of M, which is held

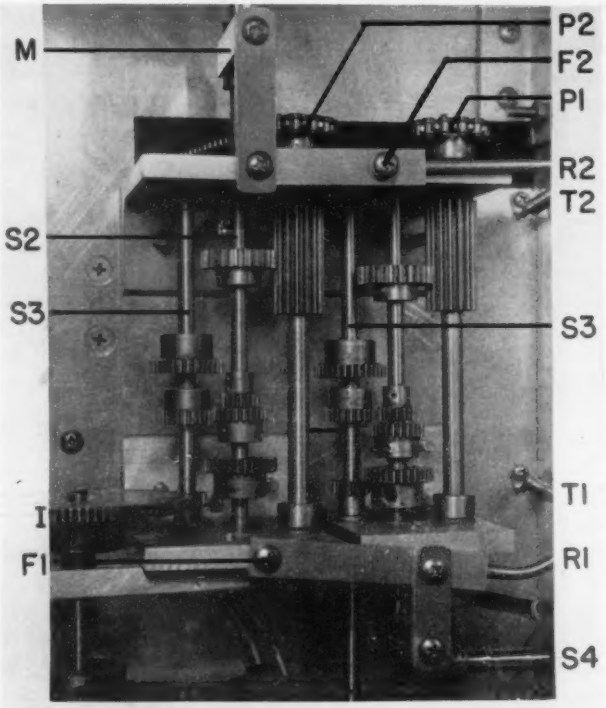

FIG. 6

in position on S2 by means of collars on either side of M. A similar mechanism positions the gear-shift idler shaft on the gear mechanism on the right. Shaft S4 (in the lower right-hand corner) is the distal end of the pinion wire which

propels the audiogram carriage board. The spur gear (32 pitch, 48 teeth) on this end of the pinion wire is concealed by the gear shift mechanism. This gear is visible in Fig. 7.

Fig. 7 shows the gear assembly for coordinating the speed of rotation of the pinion wire and the oscillator drive. All the gears in this assembly are 32 pitch gears. S3 is an extension of the right-hand S3 of Fig. 6. B is a bearing to support

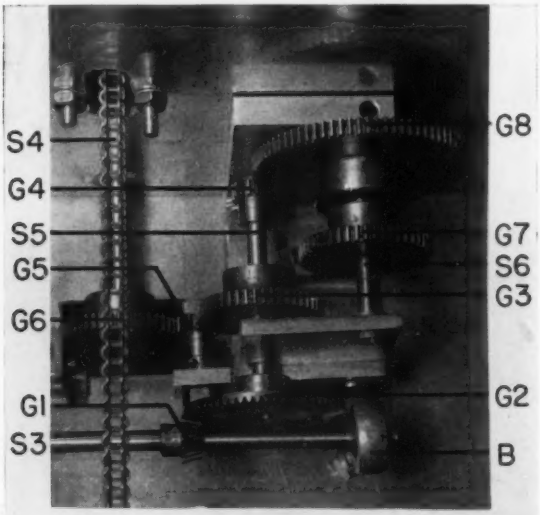

FIG. 7

the end of S3. G1 (16 teeth) and G2 (64 teeth) are bevel gears. G2 drives shaft S5, on which are attached G3 (48 teeth) and G4 (16 teeth). The gears on S5 (G4 and G5) drive both the pinion wire which propels the audiogram carriage board and the mechanism which varies the frequency of the oscillator. G3 drives the idler G5 (16 teeth), which in turn drives G6 (48 teeth) which is attached to S4, the distal end of the pinion wire. G5 may or may not be necessary, depending upon the direction of rotation of the oscillator frequency

changing dial to increase the frequency level generated by the oscillator. Attached to shaft S6, which drives the oscillator frequency changing mechanism, are G7 (48 teeth) and G8 (80 teeth). The position of S6 can be shifted to obtain either of two rotation speeds or to a neutral position. With G4 and G8 meshed as shown, the slowest speed of rotation of S6 results, which makes possible the octave spread feature. When S6 is pushed inward from the control panel as far as possible the speed is increased by a factor of five. S6 has a middle neutral position which enables the oscillator to be set at any given frequency level for a test at that level over a time interval as great as 20 minutes if desired. When the gear-shift idler shaft of the mechanism which drives S3 is in a neutral position (see Fig. 6) and G3 is meshed with G7 (see Fig. 7), manual rotation of the pinion wire by turning the knob on the proximal end of the pinion wire (see Fig. 1) synchronizes the positions of the audiogram blank and the frequency level generated by the oscillator for resetting the instrument to its original starting point or for retesting any part of the test range desired. S6 (see Fig. 7) is held in any one of its three positions by means of a ball bearing detent and three appropriately spaced V-shaped grooves cut in the circumference of the shaft. The hold which can be seen in the top of the bearing plate just over the top of G8 contains a 3/16-inch diameter ball bearing, short length of 3/16-inch compression spring and a ¼-inch diameter set screw, which comprise the detent. S6 extends through the control panel, a sprocket and a knob being attached to this external shaft extension. This sprocket and knob can be seen just to the left of the distal end of the pinion wire in Figs. 1 and 4. The speed of rotation of S6, the pitch diameter of the sprocket on S6 and the pitch diameter of the driven sprocket and gears on the oscillator frequency changing dial collectively determine the rate of frequency change of the oscillator.

The instantly reversible motor which drives the attenuator gear-shift mechanism has a shaft speed of 4 r.p.m. in either the clockwise or the counter-clockwise direction. The motor which drives the synchronized oscillator frequency changing

and audiogram carriage board mechanisms has a shaft speed of 1 r.p.m. If this motor was of the reversible type the acuity test could be given from the low to the high frequency levels in the conventional manner and then immediately repeated with the frequency decreasing from the high to the low frequency levels if desired.

The audiometer is provided with several automatic safety features. Each motor is supplied with a separate "ON-OFF" toggle switch. A pin extending at right angles from the flat side of the ladder chain sprocket on the attenuator shaft trips either of the two microswitches when the shaft has been rotated to its extreme setting in either direction. These microswitches are connected in the electric circuit of the instantly reversible motor so that the direction of the shaft rotation is reversed a few degrees when the limit positions are reached. The microswitches also limit the extreme excursion of the recording pen in either direction.

A third microswitch is mounted under the top aluminum sheet in such a way that the leaf of the switch extends through the sheet and along the right-hand side of the pinion wire near its middle. A piece of angle iron mounted under and near the right end of the audiogram carriage board (see Fig. 8) trips the microswitch and stops the motor which drives the pinion wire and oscillator frequency changing dial when the test is completed. Two push buttons (see front right-hand corner of Fig. 1) also are connected in the instantly reversible motor circuit so that the operator, by pushing the correct button, can cause the intensity level of the tone either to increase or decrease whenever desired, regardless of how the patient is using his signal key. These buttons also enable the operator to set the intensity level at the subthreshold or other value before beginning the test.

The first step in the calibration procedure is to obtain threshold acuity curves on several theoretically normal ears while the audiogram carriage board is driven in a straight line from right to left. To insure the board keeping a constant straight line position, a ball bearing three-eighths of an

inch in diameter is mounted on a rigid shaft near the middle and just to the left of the pinion wire (see Fig. 8). A rectangular slot having a width slightly greater than the diameter of the ball bearing and a length in excess of the width of the audiogram blank is cut in a metal sheet which is then fastened to the underside of the audiogram carriage board. (This straight slot is not shown in any of the illustrations.)

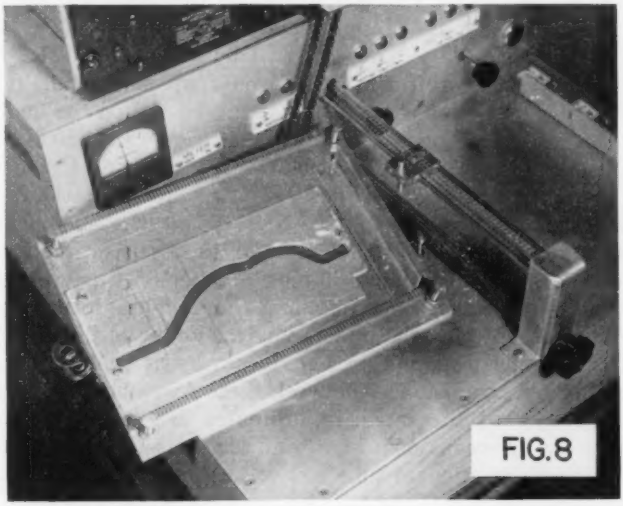

FIG. 8

The ball bearing fits into this slot when the board is placed in position on the pinion wire. The "average" or "normal" threshold acuity curve obtained with the straight slot is a concave curve (looking at it from the bottom of the audiogram), the exact shape of which is a function of the sensitivity of the ear at different frequency levels, the response characteristics of the earphone used for the calibration, and the frequency discrimination (nonlinear distortion) of the oscillator and the attenuator. When the shape of the "average" or "normal" threshold acuity curve is determined by use of the straight slot, the slope or contour of this curve is cut in a

metal sheet which is then fastened underneath the audiogram carriage board with the convex aspect of the curve presented toward the bottom of the audiogram blank (see Fig. 8). The calibrated audiogram carriage board is placed on the pinion wire so that the slot of the curve engages the ball bearing, which exerts sufficient pressure against the slot to force the board to shift along its Y-axis as it is propelled by the pinion wire. If the "correction" curve has the correct slope, the mean of the intensity ranges about the threshold will appear on the audiogram as an approximately straight line (see Fig. 2). It is advisable to have a board with a straight slot for use in testing recruitment at a fixed frequency level. It is possible to fix both a straight and a curved slot on the underside of the same board. The pen carriage mechanism can readily be shifted to any position on the chain and tightened firmly at the correct "zero" position after the calibration procedure has been completed (see Figs. 1 and 8). The correct spacings on the audiogram blank for the desired frequency abscissae are obtained by clipping a sheet of paper on the audiogram carriage board, meshing the oscillator and pinion wire drives, rotating the pinion wire manually until the oscillator dial indicates the desired frequency levels, and recording with the pen the distances between these frequency abscissae. The spacing of the intensity ordinates can be determined accurately by means of a high gain low noise level amplifier and a sensitive vacuum tube a.c. voltmeter.

Bekesy suggests the incorporation of a controlled variable intensity masking noise in the instrument. This may be done by synchronizing the rotation of the shafts of the test tone attenuator and the masking noise attenuator so that the intensity of the masking noise increases or decreases as the intensity of the test tone increases or decreases. This minimizes the possibility that the intensity level of the masking noise will become sufficiently high to mask or interfere with the acuity of the ear under test due to any "shadow curve" effect of the masking noise. Bekesy also describes a method whereby the same attenuator can be used for both the test tone and the noise simultaneously by imposing direct current

on the audio current or test tone. "The attenuated direct current can then be separated from the audio frequencies by a filter and interrupted to furnish the masking noise."[1]

Permaflux or equivalent or better quality dynamic earphones should be used with the instrument. Microswitches make satisfactory patient signal keys. Additional modifications of the component parts will produce desirable improvements in the performance of the instrument. An oscillator which can produce a frequency warble of desired characteristics may be preferable to the conventional signal generator. A reversible motor drive of the oscillator-pinion wire mechanism will permit the test to be given with either increasing or decreasing frequency change. Other calibration mechanisms might be an improvement in certain respects, and other types of drive and speed changing schemes may possess definite advantages. The instrument in our clinic is revised quite often.

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TREATMENT OF HAY FEVER, VASOMOTOR RHINITIS AND VASOMOTOR SINUSITIS WITH SODIUM PSYLLIATE—900 CASES.

JOHN H. CHILDREY, M.D.,
San Jose, Calif.

In otolaryngology, allergic conditions are among the most common requiring treatment. When the pathologic process involves the nasal mucosa, the accessibility of the latter offers advantages in treatment not often present in allergic conditions affecting other portions of the body. Submucosal injections of a 5 per cent solution of sodium psylliate intranasally is an effective therapeutic procedure offering certain advantages over other intranasal and other antiallergic treatments at present. This report on 900 allergic patients so treated in the past seven years, with follow-up data on 568 (63 per cent) unselected patients may be of interest to those who encounter some of the many manifestations of allergy.

Since intranasal vasomotor instability rather than infection is the basis for the symptoms and pathology of nasal allergy, it is suggested that the term vasomotor rhinorrhea is preferable to that of vasomotor rhinitis. The term hay fever, whether seasonal or perennial, may be used correctly in describing only a proportion of cases of nasal allergy and should be used less freely.

The average age of patients treated was 32.2 years; they were seen from the age of one to well in the seventies. Nine and four-tenths per cent were less than 10 years of age, 45 per cent were in the third and fourth decades, and there were 8 per cent more males than females.

The group treated from 1944 to 1951 comprised 11.4 per cent of all the patients examined. Since many did not elect

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to have treatment, it is evident that the incidence of allergy in otolaryngology must be appreciably higher. A higher percentage (15 per cent) of patients were treated in May due to the incidence of seasonal allergy, but even in November when the lowest percentage (4.8 per cent) was examined, it was evident that the disorder is principally chronic. The chronicity is further attested by the duration of symptoms, because in only 17 per cent of patients had symptoms been noted for less than one year, while 48.9 per cent of patients stated they had had symptoms for from six to 40 years. A history of allergy in the family was common.

The patient's clinical history is an important guide to diagnosis and treatment, and symptoms were described in the following order of frequency: nasal obstruction, postnasal discharge, nasal discharge, discomfort in the throat, asthma, headache usually associated with nasal obstruction, vasomotor symptoms referred to the eye and stuffiness of the ears and other aural symptoms. It was surprising to find conduction type of deafness in 16 per cent of cases and asthma in 14.6 per cent of cases. Anosmia was common, particularly in more chronic cases. The most common symptom, that of nasal obstruction, was often associated with sneezing, itching of the nose and watery nasal discharge. Nasal obstruction, though variable, was usually worse indoors, and worse when the patient lay down, when it might be alternately on one or the other side of the nose, depending upon the position of the head. Not infrequently when there were septal spurs or deviations of the nasal septum with obstruction, both common findings, the patient definitely stated that the more open side of the nose, that is, objectively, gave much more trouble and was much more often obstructed. This indicates the importance of vasomotor irritability of the mucosa rather than septal deformities in producing nasal obstruction. On inspection of the nasal mucosa, it was usual to find it to be boggy, pale and swollen enough to produce obstructive symptoms. In cases of long standing, polypi were not uncommon. Examination of the nasal mucus for eosinophiles showed the average percentage of these to be 9.7 per cent, but their

presence could not be considered diagnostic. Although nearly all patients had had treatment previously for allergy or treatment or surgery directed to the nose or sinuses, relatively few had previously had Roentgenograms made of their sinuses. Of 323, or 35.8 per cent, of those patients receiving treatment, in whom Roentgenograms of the sinuses were made, the findings were positive in 93 per cent, and in 64.9 per cent of the latter there was reported pansinusitis, bilateral antral involvement or involvement of both antra and of both ethmoid groups of sinuses. The findings usually consisted of much or moderately thickened mucosa. The sphenoids were seldom reported to be involved. Irrigations of an antrum or of the antra were performed in 39, or 12 per cent, of the above 323 cases, and in 19 cases pus was obtained. Operations on the antrum, however, were done in only eight, or 2.4 per cent, of the 323 cases, indicating that in spite of positive Roentgenograms and of the presence of pus in an antrum, surgery is as a rule unnecessary when allergy is the underlying etiological factor. It seems more important that the irritated nasal mucosa be treated and in those patients with nasal polypi it seemed that, following their removal, if the nasal mucosa was adequately treated, the recurrence of polypi was often definitely postponed or entirely prevented.

The present management of pollen hay fever consisting of environmental control and desensitization seems most difficult and often unsatisfactory. Skin testing leaves much to be desired. Many of the patients studied had previously tried both forms of treatment. The average length of previous treatment with injections of antigens was 2.38 years. Those patients who previously had been operated upon or had previously had intranasal cauterization were 13.6 per cent, those operated upon only were 11 per cent of the total. The most commonly performed operations done previously were submucous resection of the nasal septum, according to the histories obtained.

In the group treated were 84, or 9.3 per cent of the total, had nasal or sinus surgery also, and several surgical procedures were sometimes done on one patient. Operations on

the sinuses were done 11 times, polypi were removed once in 45 cases, more than once in five cases, and submucous resections were done in 51 cases.

In small children there seems no good reason for postponing the removal of infected tonsils and adenoids, but I am sure that too often the tonsils and adenoids are blamed when actually so-called colds, sinusitis, rhinitis, bronchitis and deafness originate on the basis of nasal allergy. I have seldom seen adenoids sufficiently hypertrophied to obstruct the nasal passages nor adenoid tissue growing in the Eustachian orifices and causing deafness. Allergic swelling of the nasal mucosa with resultant sinus and bronchial pathology is a much more likely cause of trouble than are the adenoids. Treatment directed to correction of the nasal allergy, even the constant use of antihistaminics, is sure to give better results than ill-advised surgery on tonsils, adenoids or sinuses.

Treatment to relieve the irritated, boggy condition of the nasal mucosa, most often seen over the inferior turbinates, has long been practiced. The nerves from the sphenopalatine ganglia, which supply the nose and are responsible for this irritation, have been treated with various sclerosing solutions, either injected, as alcohol into the ganglion; or applied topically, as phenol, silver nitrate, trichloracetic acid, to the mucosa over the ganglia. The average duration of relief following the application topically of silver nitrate is nine months. The surgical diathermy needle is used to scarify areas in the boggy mucosa covering the inferior turbinate bones. Zinc ionization is another method of producing an intranasal reaction, following which the mucosa and its nerves were less sensitive. Injections of small quantities of soapy solution such as sodium morrhuate have given good results, and, since 1940,[1] good results with perhaps less localized reactions were obtained by the injection of a 5 per cent solution of sodium psylliate into any areas of the mucosa which appear swollen and irritated. I have found it necessary in many cases to treat the mucosa of the septum and middle turbinates as well as that of the inferior turbinates. Treatment should be conservative and controlled in order to avoid sloughing of the mucosa, and the olfactory areas are to be avoided.

The effect of topical application of sclerosing solutions on the nasal mucosa was demonstrated experimentally.[2] Silver nitrate solution, 20 per cent, applied to the mucosa in the sphenopalatine area in the nasal passages of rabbits changed both epithelium and nerve fibres, and the regenerated epithelium showed marked cellular infiltration, while the nerve fibres showed this change and also vacuolization.

The advantage of the use of sodium psylliate over the other measures mentioned appear to be in the longer periods of relief obtained, and the greater ease in administering the solution than, for example, the use intranasally of the surgical diathermy needle. Where the use of sclerosing solutions topically can be avoided, there seems also to be less chance of sloughing of the mucosa. The morrhuate solution seemed to produce more swelling and pain than followed the use of sodium psylliate.

In allergic children, the injections of sodium psylliate are given with the patient under general anesthesia, not infrequently at the time of removal of tonsils and adenoids, while in adults local anesthesia is adequate for the intranasal procedure, 2 per cent pontocaine being used topically.

Extreme care is taken to avoid injecting the solution into a blood vessel. In order to minimize the post-treatment discomfort, caused by swelling of the mucosa, the sodium psylliate is usually mixed with 1 to 4 per cent procaine in a proportion of three to four parts psylliate to one part procaine. Discomfort following treatment is usually limited to nasal obstruction caused by swelling of the mucosa, but may be associated with swelling of the soft tissues of the nose and lower eyelids. Very rarely a temporary shingle-like reaction involved the palatal mucosa supplied by the nerves from the sphenopalatine ganglion.

The average total quantity of solution injected per patient and per area injected was 0.23 cc. to one side of the septum, 0.08 cc. to the middle turbinate of one side, and 1.87 cc. to the inferior turbinate of one side. One to two weeks were allowed to elapse between treatments, although not infre-

quently some patients received treatment to both sides of the nose, except the septum, at one sitting. Patients considered unsuitable for treatment are those with severe asthma, and in these cases, if the nose was treated, it was necessary to explain that while the nasal treatment might result in some relief of asthma, this should not be expected, as the treatment was directed to relieve only the nasal, sinus, ear and eye symptoms. Others considered unsuitable for treatment were patients with nasal allergy but with such unstable nervous systems and usually with such a multiplicity of other complaints that psychiatric rather than intranasal treatment seemed advisable.

Following intranasal treatment with psylliate, some relief of symptoms could be expected in two or three weeks. Within a few months after treatment, the mucosa, formerly pale, greatly swollen and boggy, is found to be firm, pink and of normal thickness, lying snugly against the bone of the underlying turbinate, and without ulceration, crusts, scabs, or atrophic areas. The sense of smell is not impaired.

The number of visits for psylliate treatments were one visit in 55 per cent cases, two visits in 26 per cent cases, three visits in 12.2 per cent; four, five or six visits in the remainder. In children under a general anesthetic when both sides of the nose could be treated, it was very rare (two or three cases) that more than one treatment was needed.

Results of treatment were shown by the replies to follow-up inquiries obtained in 568 cases treated. Seventy-two patients (12.6 per cent) reported no benefit (average 4.33 cc. solution used per patient), while 496 (87.3 per cent) reported that the treatment helped them. Of the latter, 50 (8.8 per cent) were better for from four to 12 months, 186 (32.7 per cent) were better for one to two years, while 260 (45.7 per cent) stated they were relieved for from two to seven years. In the latter group, an average of 5.09 cc. of solution was used per patient.

Of these 260, relief was as follows: two years in 63 cases, three years in 60 cases, four years in 47 cases, five years in 41 cases, six years in 40 cases, and seven years in nine cases.

In 77 cases, patients returned for further treatments after intervals of from one to seven years, and of these five (6.4 per cent stated they were not helped, while the remaining 72 reported benefit, of which group, 54 (70.1 per cent) reported benefit lasting from two to six years. The average quantity of psylliate solution per patient was 8.02 cc. in this group.

The average total quantity of procaine-sodium psylliate mixture used in all cases was 4.36 cc. In 260 patients reporting most benefit, the total average quantity of solution used was 5.09 cc. per patient. In 35 more than usually severe cases of vasomotor rhinorrhea in adults, the total average quantity of solution used in several treatments was 11.9 cc. Of these, 26, or 74 per cent, reported much help, four reported moderate (one to two years) relief, while five were helped little (four to 12 months).

In many adults relief of anosmia was a common result, while in children with conduction deafness improvement in hearing was the rule.

Of 12 patients with asthma, eight volunteered that the intranasal treatment cured the asthma, while four stated their asthma was much better. Of the 72 patients reporting no benefit from treatment, 16 (22.2 per cent) were asthmatic, as compared with 14.6 per cent asthmatics in the whole group treated.

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OSTEOMYELITIS OF THE FRONTAL BONE.*

ALVIN B. H. MIRMELSTEIN, M.D.,
Newport News, Va.

Osteomyelitis of the frontal bone complicating frontal sinusitis is today, fortunately, only occasionally encountered. The widespread use of chemotherapy and the antibiotics in addition to a better understanding of nasal physiology and allergic manifestations and management have, for the most part, been the major factors in bringing this about.

The condition was, and continues to be, terrifying to both patient and physician. As brought out by Brown,[1] it "presents difficulties in early diagnosis, taxes the surgeon's resourcefulness in making a decision to operate, frightens the patient with the possibilities of the resulting deformity, and forms the basis for unlimited worry and anxiety on the part of the patient's family."

Since the appearance of Mosher's[2] comprehensive paper in 1940, the types of osteomyelitis, the anatomy and histology of the diploetic veins and bone marrow, and the complications have not changed, but, as he rightfully predicts in his concluding paragraph, the advent of modern drugs as we know them has altered in many ways the treatment and prognosis, and has masked the signs and symptoms formerly found in the natural course of the disease.

There are three distinct types of osteomyelitis: fulminating, acute and chronic. The fulminating type, formerly the most dreaded of the three, is rapidly diminishing under conservative present-day management; however, it should be remembered that the more localized types in some instances may suddenly progress rapidly and have a fulminating

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course. We shall concern ourselves primarily with the acute — which may occur spontaneously, complicating sinus infections or following surgery, and the chronic — which may be a quiet continuation of acute, or cases of acute which have been improperly or inadequately treated.

ETIOLOGY.

Acute: Pus under pressure in a sinus which is not draining is the main etiological factor. Blockage may be due to allergies, post-traumatic deformities, nasal polypi or enlarged turbinates.

Chronic: Is primarily a quiet continuation of an acute process, or, becoming more into being today, cases of acute sinusitis which have been inadequately treated.

BACTERIOLOGY.

Acute: In a series of cases seen by MacBeth,[3] in which bacterial records were available, the causative agents found were staphylococcus aureus, 39 per cent; anaerobic streptococcus, 35 per cent; streptococcus hemolyticus, 9 per cent; staphylococcus albus, 4 per cent, and sterile, 13 per cent.

Chronic: Usually caused by the same organisms. Secondary invaders may at times cause a difference in cultures taken from the bone and the involved sinuses.

PATHOLOGY.

Acute: A thrombophlebitis of the veins of the sinus mucosa extends by direct spread, causing an infection of the diploic spaces. It is the tortuosity of these veins which makes removal of grossly healthy bone for a distance of 4 to 5 cm. at the time of surgery imperative. The diploic veins drain primarily towards the dura. Regenerative capacity is slight, and the suture lines of the skull have little, if any, isolating function.

Chronic: The blood supply is gradually cut off by a process of thrombosis and endarteritis. Osteogenesis and calcification

ensue. In the less chronic, sequestra are more common; in the more chronic, a fibrosis of the marrow spaces occurs with relatively smooth healing.

SYMPTOMATOLOGY.

Acute: Headache may be generalized or occipital, and usually occurs at night. Pyrexia may be present, but is variable. Frontal swelling is common. This is at first "doughy" and tender but may later become fluctuant. Proptosis is quite common.

Chronic: Frontal headache develops gradually and is not severe. Pyrexia is not usually seen. There is a slow progressive osseous swelling over the frontal area. There may be proptosis of one or both eyes.

RADIOLOGY.

Acute: There are no changes for the first seven to 10 days. Later, mottling appears, but the diagnosis should be made before radiographic evidence is positive.

Chronic: Sequestra are present in the more acute forms. Sclerosis and osteitic changes are present in the very chronic.

COMPLICATIONS.

Acute: Epidural abscess, pachymeningitis, brain abscess, acute meningitis, and thrombosis of the venous sinuses should be watched for.

Chronic: The osteitic process may spread and involve the cranial nerves. Septic thrombophlebitis may lead to meningitis or brain abscess.

TREATMENT.

Acute: The patient should receive large doses of the drugs known to be effective against the particular organism present, and drainage should be established early. An external trephine is advocated during the acute stage. Care should be taken to remove no more bone than is absolutely necessary

and spongy bone should be avoided. Therapy should be continued long after the clinical signs and symptoms have subsided. The patient should be re-X-rayed at monthly intervals for nine to 12 months.

[Treatment of the fulminating type is essentially the same, but should be more vigorous, and supplemented with oxygen, blood and other supportive measures as the need arises.]

If the lesion continues to spread, or even if localized persists, operative removal should be undertaken. The anticoagulants may have a place in preventing thrombosis.[4]

Chronic: Wide excision of the osteitic bone is the treatment of choice after the acute process, if present, has been controlled.

As mentioned previously, a margin of bone beyond the obviously diseased process should be removed.

Since the advent of chemotherapy, a primary surgical closure with adequate drainage and irrigation has been possible.

Regeneration of bone in some instances, especially in the very young, has been complete with a good cosmetic result. If not, suitable plates of tantalum have proved to be useful and completely satisfactory.

Prognosis in all cases should still be carefully guarded, and the patient should be examined frequently, both clinically and radiographically.

I should like to present two cases of osteomyelitis of the frontal bone.

CASE REPORTS.

Case 1 (Acute): H. D. R., 23-year-old white male, was admitted to the Veterans Administration Hospital on Jan. 7, 1952. Five weeks prior to admission he first had onset of coryza, which developed into frontal pain with edema and tenderness over the anterior frontal region. He was hospitalized elsewhere and was given penicillin, but this was shortly discontinued when patient proved to be sensitive to that drug. He had four X-ray treatments to the frontal area, after which the edema improved, but did not completely subside. Nasal discharge persisted, but was diminished, and he was discharged from that hospital after one week. Two weeks prior to admission the symptoms recurred, and the frontal swelling had become larger than at any time previously.

On admission there was swelling over the frontal area measuring approximately 4 cm. in diameter in the midline just above the brow line. This was extremely tender to palpation but was not fluctuant. There was a considerable amount of mucopurulent discharge in both middle meatuses. Ewing's sign was positive bilaterally.

Blood count, urinalysis and serology were normal. X-rays of the paranasal sinuses (see Fig. 1) showed considerable clouding of the antra and frontal sinuses with some thickening of the frontal bone anterior to the frontal sinus. There was no definite evidence of osteomyelitis of the frontal bone.

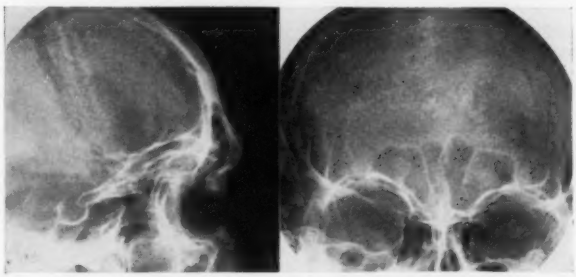

Fig. 1. Case 1. Admission Roentgenograms of frontal sinuses showing some thickening of the frontal bone anterior to the sinus and clouding of the frontal sinus.

Temperature on admission was normal. On Jan. 9, the antra were irrigated and a copious amount of thick purulent material was removed. On Jan. 10, patient had bilateral trephination of the frontal sinuses, and a large amount of pale yellow, thick, creamy discharge was released under pressure. The mucosa grossly was normal. Drains were left in place for 72 hours, at which time the discharge ceased spontaneously. Cultures revealed a nonhemolytic streptococcus which was sensitive to terramycin, streptomycin, aureomycin and chloromycetin, but resistant to penicillin (see Fig. 2). He was, therefore, given terramycin 500 mgm. every six hours and chloromycetin 500 mgm. every six hours, and treatment was continued for 21 days.

His condition remained good following trephination, and although the frontal swelling disappeared, the area for nasal drainage was not deemed adequate. Allergic studies were negative. On Jan. 22, he had bilateral antrotomies and in-fracture of the middle turbinates.

At the time of discharge patient was symptom-free. The frontal swelling had completely subsided and the areas for nasal drainage were considered adequate.

Case 2 (Chronic): J. E. K., 26-year-old white male, was admitted to the hospital on Jan. 2, 1952, complaining of intermittent frontal headache of five years' duration. Onset followed coryza and he was treated with several injections of penicillin and symptoms subsided. Postnasal discharge persisted and headaches recurred several times yearly. Three years previously he had had a bilateral nasal polypectomy, but allergic studies

were not carried out. Over a period of two years the pains had become more frequent and more severe, especially for the past three months. Swelling of the forehead had been present for approximately one year. There had been no nausea, vomiting, diplopia or vertigo. He estimated that he had received 10 injections of penicillin during this period.

On admission both middle meatuses contained a considerable quantity of thick mucopurulent discharge. The nasal mucosa was edematous and pale. A large solitary mucus polyp was present in the middle meatus, posteriorly, on the right. Ewing's sign was positive bilaterally. There was grossly diffuse swelling of the frontal area, mainly on the right. This was only slightly tender to palpation and was not fluctuant.

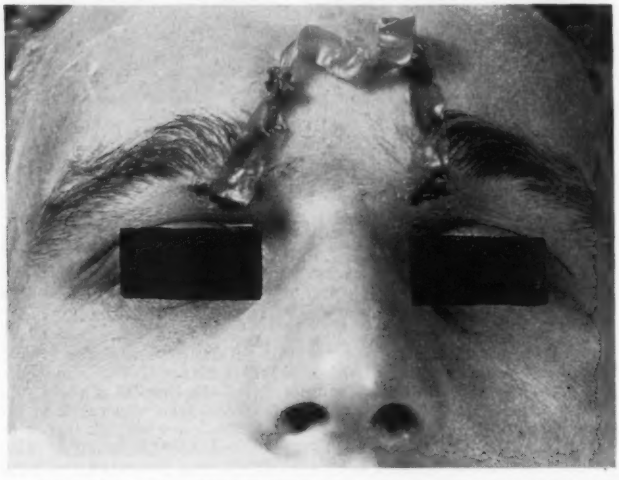

Fig. 2. Case 1. Photograph taken on first postoperative day showing position of trephines with drains in place.

Blood count, urinalysis and serology were normal. X-rays of the paranasal sinuses revealed clouding of all the sinuses with considerable bony sclerosis in the superior portion of the frontal bone (see Fig. 3). Lateral projection revealed anterior thickening of the frontal bone overlying the frontal sinuses, and the junction of the mucoperiosteal borders was very indistinct. Laminograms of the frontal sinuses revealed a chronic inflammation involving the frontal area consistent with an osteitis, but showing no evidence of osteomyelitis.

Temperature on admission was normal, and aside from frontal pain, swelling and nasal discharge, patient was asymptomatic. Antral washings revealed a copious amount of mucopurulent exudate which failed to reveal any growth. In spite of this, treatment consisting of aqueous procaine penicillin 300,000 units twice daily and terramycin 500 mgm. every six hours, in addition to local nasal shrinkage was started. On Jan. 10, bilateral trephination of the frontal sinuses was performed, releasing

thick, creamy pulsating material from both sides. Drains were left in place for 72 hours, at which time the drainage ceased. Cutures revealed anaerobic streptococci, which were greatly sensitive to terramycin, streptomycin, bacitracin, aureomycin and chloromycetin, but resistant to penicillin. Penicillin was, therefore, discontinued, and chloromycetin added as 500 mgm. every six hours.

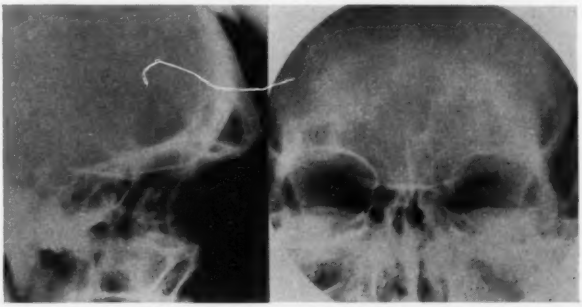

Fig. 3. Case 2. Admission Roentgenograms of frontal sinuses showing marked thickening of the frontal bone anterior to the sinus. The sinuses are cloudy and the mucoperiosteal borders are very indistinct. Sclerosis is marked.

Fig. 4. Case 2. Photograph taken on third postoperative day following external radical frontal surgery showing the catheters in place. Openings were made in that part of the catheter resting in the frontal sinus and irrigations were used both from above and below.

Allergic studies revealed marked sensitivity to dust, feathers, silk and tobacco, and precautions were taken.

On Jan. 22, bilateral antrotomies, in-fracture of the middle turbinates, nasal polypectomy and intranasal ethmoidectomy were carried out to facilitate drainage. In spite of this, headaches and discharge persisted and the forehead swelling remained. On Feb. 5, bilateral radical frontal surgery was performed, revealing a large amount of creamy pus which cultured no growth. The mucosa was edematous and grossly chronically infected. This was completely and carefully removed. The inner table was intact and grossly without defect or obvious involvement. A large encapsulated ethmoid cell containing thick exudate under pressure was found just inferior to the frontal sinuses in the midline. This was completely removed, so that the frontal sinuses were united. Bone overlying the right frontal sinus was pitted and friable, and a section was sent to the laboratory for microscopic diagnosis. Drains were left in place for four days and the sinuses were irrigated daily (see Fig. 4).

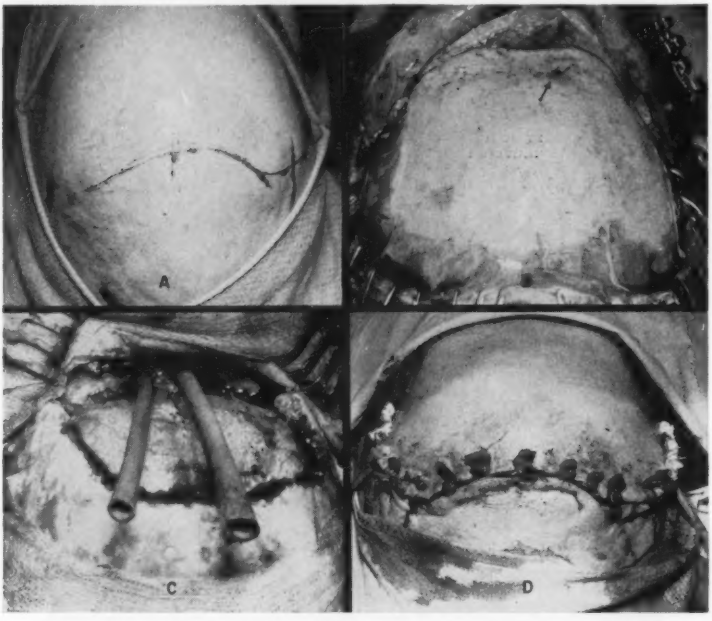

Fig. 5. Case 2. Photographs taken at the time of the excision of the frontal bone. (A) Modified coronal incision immediately posterior to the hair line. (B) Flap drawn anteriorly revealing small fistula tract in bone on right (arrow). (C) Diseased bone with suitable margin has been removed and large rubber catheters left in place extending from epidural space downward through nasofrontal ducts. (D) Incision closed. Transepidural catheter in place shown laterally.

Following surgery, the patient did very well for awhile, but on the twelfth postoperative day his temperature began to spike daily to 100°. Bone sections previously obtained revealed focal osteomyelitis and radical removal of the frontal bone was deemed advisable.

On March 6, a radical excision of the frontal bone with saucerization and curettage of the frontal sinuses was carried out. A modified coronal incision was used immediately posterior to the hairline, and the flap was drawn anteriorly (see Fig. 5). A small fistula in the frontal bone was present on the right. Sequestration was not present. The bone was ebonated, but the underlying dura was normal. All grossly involved bone was removed, and a margin of approximately 4 cm. of apparently normal bone was similarly removed. The frontal sinuses were found to be in surprisingly good condition, but the inner plate was nevertheless removed. Most of the superciliary ridge was preserved. Two No. 20 perforated rubber catheters were left in place, extending from the epidural space downward through the nasofrontal duct, and one No. 16 transepidural catheter for irrigation with bacitracin was left in place. The incision was closed. Convalescence was uneventful. The drains were removed on the seventh postoperative day, and headaches, discharge and swelling have not recurred. Antibiotics were continued for 28 days. X-rays on March 25 revealed normal bone surrounding the surgical defect (see Fig. 6). To date, there have been no complications, and impressions have been made for a tantalum plate which will be inserted later.

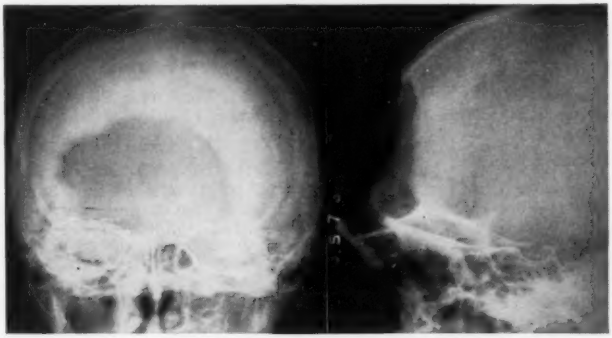

Fig. 6. Case 2. Roentgenograms showing normal bone surrounding surgical defect.

CONCLUSION.

In conclusion, one fact of great importance should be emphasized. It is becoming more and more evident that penicillin is becoming less and less effective in the treatment of infections formerly very susceptible to this drug. Davison[5] reports that in 1946, 82 per cent of all gram positive cocci

isolated from patients with infections of the paranasal sinuses were penicillin sensitive. In 1949, this number had decreased to 12 per cent and is today probably even further diminished. He postulates that perhaps the routine use of penicillin in infections of this type has set the stage for more virulent organisms proving to be penicillin-fast. Local sprays, ineffective doses and disregard of physiological principles also may be possible reasons for this (Goodale[6]). The physician should not have a false sense of security and feel that drugs alone will eliminate paranasal sinus infections. Cultures should be carried out, and these should include anaerobic studies. Drug sensitivities also must be carried out if these diseases are to be properly treated and eliminated. Anything short of this might well lead to a reappearance of all types of complications, not only of paranasal sinus infections but of all the infections peculiar to the ears, nose and throat.

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December 1, 1952.

Audicon Models 400 and 415.

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Auditone Model 11.

Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67.

Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3; Dahlberg Model D-4.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 149 Church St., New York 7, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.

Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis 1, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone T5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Normatone Model C.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co.

Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3.

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp.

Rochester Model R-1; Rochester Model R-2.

Manufacturer: Rochester Acoustical Laboratories, Inc., 117 Fourth St., S.W., Rochester, Minn.

Silvertone Model J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal; Model Cameo.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 64 S. Bonnie Brae St., Los Angeles 5, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Albert C. Furstenberg, University Hospital, Ann Arbor, Mich.
Vice-President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Me.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Meeting: Roosevelt Hotel, New Orleans, La., May 1-2, 1953.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Roosevelt Hotel, New Orleans, La., Apr. 26-27, 1953.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. F. E. LeJeune, Ochsner Clinic, New Orleans, La.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Roosevelt Hotel, New Orleans, La., April 28-29, 1953 (mornings only).

MID-WINTER.

Triological Council Meeting—Hotel Syracuse, Syracuse, N. Y., 9 a.m., Jan. 6, 1953.

Eastern Section Meeting—Hotel Syracuse, Syracuse, N. Y., Jan. 7, 1953.

Southern Section Meeting—Andrew Jackson Hotel, Nashville, Tenn., Jan. 12, 1953.

Middle Section Meeting—The Drake, Chicago, Ill., Jan. 19, 1953.

Western Section Meeting—The Elks Club, Los Angeles, Calif., Jan. 24, 1953.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Carl H. McCaskey, 608 Guaranty Bldg., Indianapolis, Ind.
Vice-Chairman: Dr. Fred W. Dixon, Rose Bldg., Cleveland, Ohio.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James M. Robb, Detroit, Mich.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting:

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., Oct. 6-10, 1952.
Meeting: Roosevelt Hotel, New Orleans, La., Apr. 21-25, 1953.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Herman J. Moersch.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.
Meeting: Roosevelt Hotel, New Orleans, La., Apr. 28-29, 1953 (afternoons only).

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Frederick Thorlakson, Cobb Bldg., Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Victor Alfaro.
Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: Edgewater Gulf Hotel, Edgewater, Miss., May 11, 1953.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Philip E. Meltzer, 20 Charlesgate West, Boston, Mass.
Secretary: Dr. Theo. E. Walsh, 640 S. Kingshighway, St. Louis 10, Mo.
Meeting:

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.
President-Elect: Dr. Kenneth L. Craft, Indianapolis, Ind.
Secretary-Treasurer: Dr. Joseph Hampsey, 806 May Bldg., Pittsburgh 22, Pa.
Meeting:

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Justo M. Alonzo, Montevideo.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: January, 1954, Mexico City.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Daniel L. Sexton, St. Louis, Mo.
President-Elect: Dr. John I. Marker, Davenport, Iowa.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.
Meeting:

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. D. A. Morgan, Norfolk, Va.
Secretary-Treasurer: Dr. G. Slaughter Fitz-Hugh, Charlottesville, Va.
Fall Meeting: For Ophthalmology, Charlottesville, Va., Nov. 18-19, 1952.
For Otolaryngology, Charlottesville, Va., Nov. 21-22, 1952.
Spring Meeting: Hot Springs, Va., May, 1953.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Victor Goodhill.
Secretary-Treasurer: Dr. Orwyn Ellis.
Chairman of E. N. T. Section: Dr. Harold Owens.
Recording Secretary, E. N. T. Section: Dr. Donald B. Hull.
Chairman of Eye Section: Dr. Deane Hartman.
Recording Secretary, Eye Section: Dr. Robert Norene.
Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

THIRD LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

President: Dr. Franz Conde Jahn.
Vice-Presidents: Drs. Julio Garcia Alvarez, Angel Bustillos and Celis Perez.
Secretary General: Dr. Victorino Marquez Reveron.
Secretary of Assemblies: Dr. Cesar Rodriquez.
Time and Place: Caracas, Venezuela, July 31, 1954.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Norman N. Smith, 291 Whitney Ave., New Haven 11, Conn.
Secretary: Dr. Joseph G. Gilbert, 111 E. 61st St., New York 21, N. Y.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. MacLean B. Leath, High Point, N. C.
Secretary and Treasurer: Dr. Geo. B. Ferguson, Durham, N. C.
Meeting:

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. C. Allen Dickey, 450 Sutter St., San Francisco, Calif.
Secretary-Treasurer: Dr. Howard P. House, 1136 W. Sixth St., Los Angeles 17, Calif.
Meeting: Los Angeles, Calif., 1953.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

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Program Chairmen:
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Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.
Mid-Winter Clinical Convention annually the last two weeks in January at Los Angeles, Calif.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

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Vice-President: Dr. Harry P. Schenck.
Treasurer: Dr. William J. Hitschler.
Secretary: Dr. John J. O'Keefe.
Executive Committee: Dr. Valentine M. Miller, Dr. C. L. Jackson, Dr. George L. Whelan.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Vice-Chairman: Dr. K. W. Cosgrove, 111 E. Capitol Ave., Little Rock, Ark.
Secretary: Dr. F. A. Holden, Medical Arts Bldg., Baltimore, Md.
Meeting: Miami, Fla., Nov. 10-13, 1952.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Melvin W. McGehee, 425 Eleventh St., Huntington 1, W. Va.
President-Elect: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.
Meeting: Hot Springs, Va., May 2-3, 1953.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. D. E. S. Wishart, 170 St. George St., Toronto, Ontario.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Minaki Lodge, Minaki, Ontario.
Time: June 14-18, 1953.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Victor M. Noubleau.
Secretary: Dr. Héctor R. Silva.
1o. Vocal: Dr. Salvador Mixco Pinto.
2o. Vocal: Dr. Daniel Alfredo Alfaro.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

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Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

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Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
Secretario Tesorero: Dr. Vicente Carri.
Sub-Secretario Tesorero: Dr. José D. Suberviola.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

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Vice-Presidente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Sec. de Actas: Dr. Juan Berini.

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Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Joao Penido Burnier.
First Secretary: Dr. Gabriel Porto.
Second Secretary: Dr. Roberto Barbosa.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. F. J. Monteiro Sales and Dr. Jose Martins Rocha.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Díaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asís R., Dr. Jorge Bergallo Yofre.

BUENOS AIRES CLUB OTORINOLARINGOLOGICO.

Presidente: Dr. Alberto P. Haedo.
Vice-Presidente: Dr. V. R. Carri.
Secretario: Dr. Renoto Segre.
Pro-Secretario: Dr. Carlos A. Gutierrez.
Tesorero: Dr. J. M. Tato.
Pro-Tesorero: Dr. Norberto Von Soubiron.

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

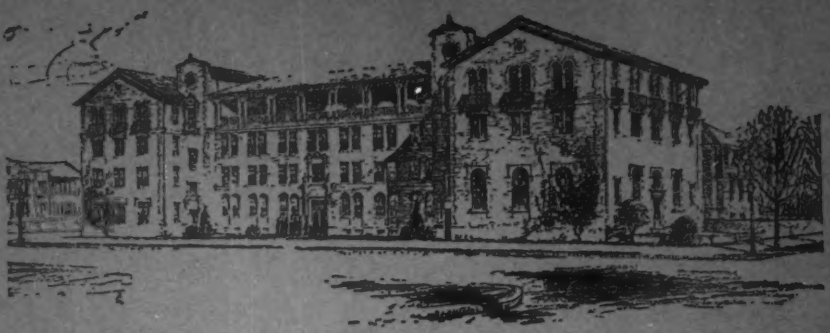
Presidente: Dr. Carlos A. Cleves C.
Vice-Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Elpidio Posada.
Tresoreo: Dr. Mario Arenas A.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.
Meeting: Dec. 12-13, 1952, San Salvador.



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